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The Massachusetts Medical Society

The next annual meeting of the Massachusetts Medical Society will be held in Boston, Monday, Tuesday and Wednesday, June 8, 9 and 10, 1931.

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BULLET IN THE HEART

Report of Case with Recovery*

BY ROBERT SHOEMAKER, 3D, M.D.,† AND JOHN C. ECKELS, M.D.†

A WHITE woman, age 23, was brought into the surgical dispensary by police ambulance at 11:15 p. m. on August 9, 1929 with two bullet wounds in her chest.

CLINICAL EXAMINATION: Lying on the stretcher, she appeared to be in extremis. Although practically unconscious, she was very restless, throwing her arms about, moving her head, and uttering moans and groans. Her color was the characteristic ante mortem grayish pallor of shock. No radial pulse was perceptible in either wrist. Examination disclosed two bullet wounds of entrance in the left chest. (Fig. No. 1.) Each was surrounded by a powder

FIG. 1. Schematic drawing showing location of bullet wounds in chest wall.

burn, which was sufficient proof that the shots had been fired at close range. No wounds of exit could be found. The left side of the thorax was hyperresonant; no breath sounds could be heard. The heart was displaced to the right and the heart sounds were best heard to the right of the sternum in the third and fourth interspaces. The sounds were faint and irregular, the irregularity consisting of a marked variation in the force and rhythm of the beats. The patient's skin was cool and moist.

TREATMENT: Emergency treatment for shock was immediately instituted. Caffein sodium benzoate 0.5 (gr. vijss) and morphine sulphate 0.015 (gr. ¼) were promptly administered, the foot of the stretcher elevated fifteen inches, and the patient covered with blankets and surrounded with hot water bottles. Ten minutes later another dose of morphine sulphate 0.015 was given together with 0.5 c.c. of adrenalin hydrochloride 1:1000. The patient showed a rapid response to treatment. Within fifteen minutes the radial pulse became perceptible and within a half hour the face had lost its ashen hue. The skin was still cool and moist. The patient was transferred to bed in a semi-comatose condition, the result partly of shock and partly of morphine. The pupils were contracted and the respirations thirty-eight per minute. The pulse was weak and irregular and could not be counted with accuracy.

The following morning the patient was conscious and rational, complained of no pain, but had some difficulty in breathing. Temperature, 99.6; pulse, 104; respirations, 24. From this time she made an uneventful recovery.

X-RAY STUDIES: On the morning after admission the patient was taken to the X-ray Department in her bed, where films of the chest were exposed and the following report rendered: "Metallic foreign body 1.0 x 0.5 cm. in size lying about 3 cm. to the left of the spinal column at the level of the eleventh rib. No other foreign body detected. Fluid in left chest with displacement of heart to the right." (Fig. No. 2.)

Since two definite wounds of entrance existed in the chest wall, further X-ray examination was carried out as soon as the patient was able to undergo systematic fluoroscopy. The following report was then returned: "Small metallic foreign body, evidently a bullet, is lodged in the musculature of the right heart, and shows an excursion of 2 cm. during the cardiac cycle. Cardiac contour normal." (Figs. No. 3 and 4.) A film of the chest was then made with an exposure of one-thirtieth of a second, successfully demonstrating this bullet which had escaped detection on the slower routine films.

OPERATION: A small area of induration and tenderness could be palpated directly over the site of the first bullet found by X-ray examination. Under local anaesthesia a 22 calibre bullet was removed from beneath the skin of the left back in the situation indicated by the first x-ray report.

Discussion: The important question that presented itself after the location of the second bullet in the heart muscle was: "What is to be done about it?" The surgical chief (Dr. John B. Deaver) decided that as long as the patient was making an excellent recovery, the bullet should be allowed to remain in situ. Another question, less important to the patient and more speculative in character, was this: "How did the bullet reach the right side of the

*From the Surgical and Roentgenological Departments, Lankenau Hospital, Philadelphia, Pa.

Including a review of cases of foreign body in the heart reported in the literature of the past 14 years (1916 to 1929 inclusive).

†Shoemaker—Roentgenologist, The Lankenau Hospital, Philadelphia and The Joseph Price Memorial Hospital, Philadelphia. Eckels—House Officer, Lankenau Hospital, Philadelphia 1928-1930. Assistant Resident in Urology, Peter Bent Brigham Hospital, 1930-. For records and addresses of authors see "This Week's Issue", page 239.

heart? What route did it take?" The wound of entrance was in the fifth interspace on the left side in the anterior axillary line. (Fig. No. 1.) To arrive at its destination, it would seem that there were three possible pathways for the bullet to pursue. First, it may have ricocheted from the inner aspect of a rib or costal cartilage. Second, it may have followed an intramuscular course through the anterior walls of both left and right ventricles. Third, it may have penetrated the lower portion of the left ventricle, the interventricular septum, and the right ventricle. It is well known that small wounds of the heart muscle in certain areas may close spontaneously due to the interwoven structure of the myocardial fibres. This is further supported by a number of cases reported in the literature, abstracts of which appear below.

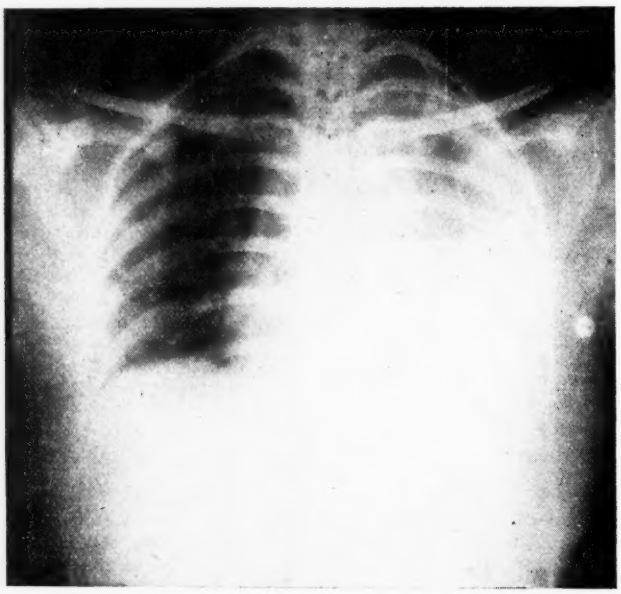

FIG. 2. Roentgenogram showing fluid in left side of thorax and displacement of heart to the right.

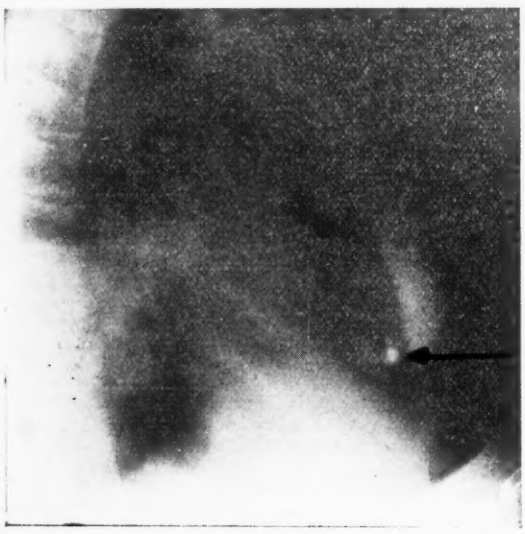

FIG. 3. Roentgenogram showing bullet in heart. Note blurring due to oscillation of bullet.

The patient was kept flat in bed for three weeks in order that no unnecessary strain might be imposed upon the heart. An electrocardiogram taken during convalescence showed a regular rhythm and a rate of 105 per minute. (Fig. No. 5.) The patient was in excellent condition upon discharge and was advised to return to our Follow-up Department once each month.

FOLLOW-UP: Patient returned at four and eight week intervals after discharge. She was a picture of perfect health, and had no cardiac or respiratory symptoms of any nature whatsoever. Fluoroscopy in

FIG. 4. Photograph of patient. Black dots indicate limits of oscillation of bullet as seen in the fluoroscope.

both instances revealed the bullet oscillating with each cycle of the heart beat. Clinically and fluoroscopically, her heart presented no abnormality in size, position, or function. An electrocardiogram was taken upon her last return. This showed a heart rate of 75 beats per minute with a slight sinus arhythmia.

REVIEW OF THE LITERATURE: The following is a brief summary of cases of foreign body in the heart which we have collected from the literature from 1916 to 1929 inclusive,—a period of 14 years. We believe that it is of sufficient importance to include a brief résumé of each case because from such a series we can form the best general plan of action in an emergency. The following abstracts are listed chronologically, as far as is possible, under each author. A complete reference may be found in the bibliography appended.

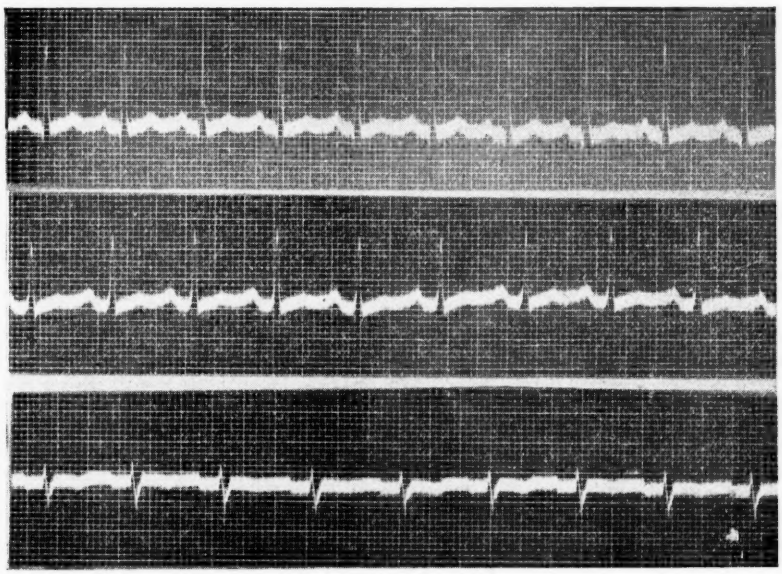

FIG. 5. Electrocardiogram made during convalescence. Resistance: 2000 Ohms. String: 1 M.V. per 1 cm. step.

1. *LERICHE*[1]—Extraction of sewing needle from the heart. Child of 9½ years fell with needles stuck in blouse. Complained of sticking pain in left breast region. Examination showed small pulsating swelling. A small object could be palpated. No cardiac symptoms. Operation several days later after local swelling had subsided. Needle located in wall of left ventricle. Muscle of left ventricle incised and needle extracted. Uneventful recovery.

2. *GRANDGERARD*[2]—A shrapnel ball entered beneath the left clavicle and was found by radioscopy in the region of the right atrium. An hour later, the ball was seen in the region of Scarpa's triangle in the course of the femoral vessels. Several minutes later, when the patient had assumed a horizontal posture, the ball was noted at the level of the right sacro-iliac synchondrosis in the course of the right iliac vessels. Operation performed and projectile found in the right iliac vein. Missile removed and vein ligated. (Ultimate outcome not mentioned. Recovery assumed.)

3. *LOBLIGEOIS*[3]—Shrapnel ball free in left ventricular cavity. No clinical symptoms. Discovered by fluoroscopy several months after injury. Foreign body moved with cardiac movements. (No reference to operation.)

4. *LYLE*[4]—Soldier wounded in right thigh. Debridement of wound. Femoral vessels exposed but no fragment found at operation or by X-ray examination. Death after 4 days and 18 hours from generalized gas bacillus infection. At post-mortem, a shell fragment 1.5 by 0.9 by 0.5 cm. with bits of clothing attached was found free in the cavity of the right ventricle.

5. *DUVERGEY*[5]—Extraction of bullet from wall of left ventricle. Gunshot wound in right lateral thoracic cavity followed by bleeding from nose and mouth. Pain in chest and severe dyspnoea. Radiograph showed bullet lodged within the heart. Four months after injury: cyanosis and persistent precordial pain. Six months after injury: fluoroscopy showed bullet in region of left ventricle; moved with cardiac movements. Seven months after injury; bullet removed by operation. Examination one month after operation: chest and heart normal; cyanosis fading.

6. *ESCANDE ET BROCQ*[6]—Chest wound below left nipple from piece of shrapnel. Little bleeding. Intense dypnoea, anguish and vertigo for 15 days. No other objective or subjective cardiac symptoms. X-ray showed a small piece of shrapnel embedded in muscle of the anterior wall of the heart approximately 3 cm. from apex. No operation. Patient living and well six months after injury.

7. *ESCANDE ET BROCQ*[6]—Projectile in wall of heart. Bullet lodged in muscle of left ventricle; syncope and mild haemorrhage. Severe dyspnoea for 4 or 5 days. Examination 17 months after injury: no symptoms; heart action and sounds normal. X-ray examination showed nose of bullet moved with respirations; base of bullet not freely movable. Examination 29 months after injury: no symptoms except slight shortness of breath on exertion and slight precordial pain. Bullet left in situ.

8. *GAUDIER*[7]—Bullet removed from heart after 3 months. Immediate effects of injury: unconsciousness for a time; pain at apex of heart; chills; fever. X-ray: bullet fixed transversely at the level of the apex of the heart; moved synchronously with left ventricular movements and independent of diaphrag-

matic excursions. Operation 3 months after injury: bullet 42 by 7 m.m. removed from anterior wall of left ventricle. Stormy convalescence. Discharged 33 days after operation.

9. *LeFORT*[8]—Extraction of projectiles from posterior aspect of heart claimed to be much more difficult than from anterior aspect. LeFort reviews briefly the following cases which are not duplicated in this series: (A.) Bullet lodged in posterior wall of right ventricle. Operation 30 months after injury with recovery. (B.) Shrapnel fragment lodged back of pulmonary vein in wall of left auricle. Operation performed, but foreign body not removed on account of symptoms of irritation of the cardiac plexus.

This author recounts five other cases of operative removal of foreign bodies lodged in, or directly posterior to, the posterior pericardium. These have been omitted because the foreign body was not within the heart proper.

10. *PEZZI*[9]—Projectile in the heart. (This case previously reported by Ascoli. Reference not given and not found.) Shrapnel wound in back followed by moderate fever, sense of pressure in epigastrium several hours after meals. Occasional acute epigastric distress; urinary retention. Fluoroscopic examination 13 days after injury showed bullet in region of left iliac crest. A second examination 44 days after injury showed no trace of bullet in former location. Bullet observed in extreme right portion of cardiac shadow; it moved with cardiac pulsation and was believed to be in cavity of right atrium. No operation. Patient discharged 12 months after injury. Heart normal in size and shape. No cardiac symptoms. Foreign body believed to have penetrated the left iliac vein and to have travelled thence to the heart via the inferior vena cava.

11. *LeFORT*[11]—Extraction of projectile from heart. War injury. Immediate effect: bleeding from nose and mouth; no loss of consciousness. Three months later: marked dyspnoea, cough, progressive auricular bruit. Fluoroscopic examination: foreign body, size of pea, in left auricle. Operation 3 months after injury. Bullet found in posterior inferior wall of left auricle. Sudden death on fourth day.

12. *PISANO*[12]—Shrapnel ball in right heart. Entrance wound in region of left scapula. Immediate effect: small pulse; severe dyspnoea; pneumothorax. Exploratory puncture of left chest several hours after injury yielded nothing. X-ray on twenty-ninth day: heart displaced slightly to the right; foreign body observed at level of hilus of left lung. Exploratory thoracentesis: one litre of blood obtained; entrance of air. Second X-ray (date not given): the foreign body had migrated to the right heart and was pressing on the posterior wall of the ventricle. Death on thirty-fourth day after injury, caused by septicaemia from wound infection.

13. *HARTMANN*[13]—Extraction of bullet from heart wall. Machine gun bullet imbedded in wall of right ventricle. Foreign body gave rise to symptoms 6 months after injury—precordial pain, palpitation and temporary arrest of heart action with sensation of imminent death, most pronounced at night. Bullet removed with immediate relief of symptoms. Patient well 2 years and 9 months after injury.

14. *McCARTNEY AND DRUMMOND*[14]—Gunshot wound of liver. Irregular piece of shrapnel one inch in length entered chest in right midaxillary line in 7th interspace; penetrated pleura, diaphragm, right lobe of liver and right branch of hepatic vein; thence carried via inferior vena cava to right ventricle. Patient had other serious wounds. Died of haemorrhage and shock 36 hours after injury. Foreign body in heart not suspected; found at autopsy.

15. *MENUET*[15]—Projectile in right ventricle. Missile entered in right axillary line at level of seventh rib. Clinically, cardiac size and movements normal; diaphragmatic excursion limited; slight dyspnoea; no cardiac symptoms. X-ray; shadow of foreign body slightly to right of cardiac apex. Death on second day following operation for leg injuries. Post mortem revealed bullet at extreme apex inside right ventricle. Bullet had entered circulation at junction of hepatic vein and inferior vena cava. Small retroperitoneal haematoma. (Note: this case is almost identical with the one reported by McCartney and Drummond[14]. We have checked these reports carefully and believe them to be separate cases.)

16. *DESPLAS ET CHEVALIER*[16]—Foreign body in anterior wall of heart. Bullet entered left ventricle and lodged in anterior wall. Enormous haemorrhage (3 litres). No operation. Died three hours after injury. Post mortem showed that bullet could have been easily recovered if operation had been performed at once.

17. *CHARPY*[17]—This author cites the following two cases previously reported: (1) Maffi[10]—Soldier received wound at the base of the right chest. X-ray showed a fracture of the right tenth rib and a foreign body oscillating within the heart shadow. Death 4 days after injury. Post mortem showed bullet had migrated to the bifurcation of the right pulmonary artery. Wound of entrance to circulatory system at junction of hepatic vein and inferior vena cava. (2) Pezzi—same case as reference No. 9.

18. *LeFORT*[18]—Projectile in left ventricle. Demonstrated first case in France in which a scrap of shell 8 by 4 by 4 m.m was successfully removed from the cavity of the left ventricle. Previously reported successful extraction of projectile from left auricle. This was the eleventh projectile he had extracted from the heart in 9 cases. One patient died on fourth day[11]; all others recovered.

19. *PONZIO*[19]—Projectile free in right ventricle. Shrapnel ball entered tangentially to iliac crest and apparently lodged against wall of large vein. Eroded through wall and was carried to right ventricle. Heart symptoms marked its passage through the right auricle, but after reaching ventricle it caused no further disturbances. Left in situ. Duration, one year after injury.

20. *COPE*[21]—Sewing needle accidentally thrust into chest wall at upper margin of fourth costal cartilage, 1 inch to left of sternum. Located by fluoroscopy, presumably in right ventricle. Operation under fluoroscopic control a few hours after accident. On opening pleura, heart fell away and swayed violently but soon resumed normal action. End of needle protruding from pericardium on the left was grasped and extracted. Recovery.

21. *LUCKETT*[22]—Removal of missile from wall of right ventricle. .32 calibre bullet entered 1st interspace ¾ inch to left of sternum and lodged in anterior wall of right ventricle. Removed 8 days later with recovery.

22. *HAYES*[23]—Rifle bullet in heart of patient who had been wounded in Mesopotamia in 1916. Examined in Dublin several months after injury. Bullet situated in inferior posterior wall of left ventricle midway between apex and atrioventricular groove. (No operation mentioned.) Patient in good health.

23. *KOCH*[24]—Projectile in heart. Soldier received wound in chest and abdomen. Unconscious for a few minutes. Was being transported back of lines when he fell from wagon striking left side of chest; dyspnoea disappeared as though by magic. After that, no symptoms or signs of physical disorder. Foreign body embedded in wall of left ventricle oscillating with cardiac movements. Perfectly well 5 years after injury; worked hard during entire interim. Foreign body not removed. Patient in good health.

This author cites 16 other cases,—all war injuries. These are listed under the authors re-

porting; some of them date back prior to 1916. The complete references in each case may be found in the bibliography appended to Koch's[24] article.

(1) *FREUND*—Foreign body in apex. Moved with respiration. No symptoms. No operation. Time after injury not indicated.

(2) *FIELIZ*—Foreign body in cardiac wall. No symptoms. No operation. 5 months after injury.

(3) *NIKLAS*—Wound in region of left shoulder blade. Foreign body in wall of left ventricle. No symptoms. No operation. Time after injury not indicated.

(4) *HELLER*—Bullet in left ventricular wall. Moved with cardiac pulsations and respirations. No symptoms till six months later; then intermittent dyspnoea and palpitation.

(5) *MUHSAM*—Bullet in muscle of one of the ventricles. No symptoms. No operation. Time after injury not indicated.

(6) *GLASER UND KAESTLE*—X-ray examination showed bullet in right portion of cardiac shadow. Some haemoptysis. No symptoms for one year, then pain over left chest from costal border to shoulder. Occasional dyspnoea. Clinical heart findings normal. Electrocardiogram showed abnormal ventricular action. No operation.

(7) *THOLE*—Abdominal wound. Several X-ray examinations, but no foreign body detected. No symptoms for 15 months, then a feeling of pressure below costal margin on the left. X-ray showed foreign body located in region of base of right ventricle moving with systole. Operation. Suppurative pericarditis and pleuritis. Death.

(8) *DIETRICH*—Bullet in wall of right ventricle. No cardiac symptoms for 4 weeks; then frequent syncope and pulse disturbances several times a day. X-ray showed foreign body moving with respiration. Operation with removal of bullet from right ventricle. Few drops of pus directly around bullet. Recovery.

(9) *JENCKEL*—Wound in left chest. Pulmonary symptoms only; heart negative. First x-ray negative. Second x-ray showed bullet in posterior wall of right ventricle. Symptoms: intermittent pulse and precordial pain. First operation: foreign body not found. X-ray 6 months after operation: foreign body in same location. Second operation: foreign body found in posterior wall of right ventricle. Removal. Recovery.

(10) *SCHNEIDER*—Foreign body in posterior wall of right ventricle with one end probably penetrating cardiac cavity. Marked cardiac symptoms. Operation; removal; recovery.

(11) *GRUBER*—Chest wound. No cardiac symptoms. Death several months later from intercurrent disease. Autopsy showed bullet in musculature of left ventricle without penetration into cardiac cavity.

(12) *BECKER*—Wound in right axilla. No symptoms for several weeks; then palpitation and dyspnoea. Examination 6 months after injury showed heart action normal. X-ray: pea-sized foreign body in apex of heart. No operation.

(13) *PICK*—Foreign body in right ventricle for 2½ years. No symptoms; no operation.

(14) *SCHUTZE*—Foreign body in right ventricle. No symptoms; no operation.

(15) *SCHUTZE*—Foreign body in wall of left heart. No symptoms; no operation.

(16) *KALEFELD*—Shrapnel ball in wall of left ventricle; moved with heart movements. No symptoms for several weeks; then palpitation and vertigo. Heart action and pulse normal. No indication for operation.

24. *KOCH*[24]—Shrapnel injury. Patient unconscious for 14 hours; felt weak; gradually improved. X-ray 10 weeks after injury was negative. No symptoms; worked hard. Four years and nine months after injury, complained of sense of pressure in epigastrium after eating; this impaired appetite. Became weak due to insufficient food. Physical examination, including heart, was normal. X-ray showed foreign body in right ventricle moving with cardiac movements. No operation.

25. *HAGEN-TORN*[25]—Pea-sized foreign body in wall of left auricle. Patient unconscious for 15 days after injury. Progressive pain in cardiac region interfering with work; palpitation. Apex beat neither visible nor audible. Operation 5 years and 2 months after injury. Recovery. Living and well 7 months after operation.

26. *TECON*[26]—Bullet in heart muscle for over 2 years, located at apex of left ventricle. Mild shock; profuse haemorrhage. Patient noticed a clicking sound in region of chest wound synchronous with respirations. No cardiac symptoms till 8 months later; then vague transitory pain in region of fifth and sixth left interspaces, gradually increasing in frequency and severity. Physical examination, including heart, was negative on several occasions. Increasing pain led to X-ray examination and localization of foreign body. Surgery advised, but patient (a soldier) was ordered home. He was able to climb 2,000 meters without cardiac embarrassment. No operation. Patient lost after 26 months.

27. *DAVENPORT*[27]—A 16 penny casing nail thrust into heart, severing the interventricular branches of the left coronary artery and vein. Operation with ligation of artery and vein with silk.

28. *STECKBAUER*[28]—While stooping over, a boy was shot in the buttock by a playmate with a .22 calibre bullet. Bullet traversed abdomen making approximately a dozen wounds in gastro-intestinal tract, penetrated the diaphragm and lodged in the heart muscle near the apex. Patient withstood general anaesthesia and recovered from his abdominal operation. Foreign body present in heart muscle and moved synchronously with cardiac pulsations 9 months after accident.

29. *FIORI*[29]—Needle in heart muscle, oscillating with cardiac movements. Intense dyspnoea and precordial pain; irregular pulse; bradycardia and tachycardia. Operation: foreign body 5 cm. in length found in posterior wall of right ventricle. Stormy convalescence due to pericarditis (staphylococcus albus). Patient discharged fortieth day post-operative. Living and well 9 months after operation.

30 *HUNTER, STAUB, LUNSFORD*[30]—Aluminum pipe stem thrust into chest, penetrating cavity of left ventricle. Striking absence of physical signs and symptoms at first. Then friction rub over precordium, increasing temperature and accelerated pulse. Unable to locate foreign body (aluminum) by roentgen ray. Death 10 days after accident. Post mortem showed fibrinous pleurisy and pericarditis. Possible blood stream infection not excluded.

31. *BIRT*[31]—Soldier shot in anterior chest just to left of sternum at level of fourth cartilage 4 weeks prior to admission. Wound healed. Two weeks prior to admission, he developed dyspnoea and pain about the heart. Roentgenogram showed bullet in anterior wall of right ventricle. Operation: some cloudy serous pericardial fluid encountered; foreign body not found. Wound left open for drainage and suction apparatus applied. Thirty days after operation bullet was discharged spontaneously through open wound. Patient recovered.

32. *PETERSEN*[32]—Large safety pin, straightened out and with clasp broken off, thrust into chest with suicidal intent; penetrated left atrium; demonstrated by roentgenogram. Operation 2 days after accident: end of pin found close to pericardium; whenever it was touched patient showed signs of collapse. Pin not removed. Stormy convalescence; some fever for

2 months. Eighteen months after accident patient committed suicide by throwing himself in front of a train. Autopsy showed a curved brass wire 12 cm. long transfixing the left atrium. Physical condition prior to death excellent.

COMMENT AND SUMMARY

1. A case of a bullet lodged in the myocardium is reported because of:

(a) The relative rarity of such a condition in civilian life.

(b) The rapid response to emergency treatment for shock.

(c) The difficulties encountered in locating the intracardiac bullet and the importance of fluoroscopy.

(d) The speculation on what route the bullet followed to its destination.

(e) The rapid and apparently permanent recovery without operative procedure.

2. A review of the literature discloses 49 similar cases reported during the past 14 years (1916 to 1929 inclusive). Most of these cases were war injuries.

The foreign body in each case was opaque to the X-ray with one exception,—an aluminum pipe stem. It must be noted that the fluoroscope is far superior to routine films for the localization of a rapidly oscillating foreign body, for with the fluoroscope the retina of the observer's eye corresponds to the sensitized film upon which the shadow is cast.

The heart has long been considered by both the laity and physicians as one of the most vulnerable spots in the human body, and perhaps rightly so. The 50 cases of intracardiac foreign body above described represent probably a small fraction of the total number of actual cases which have occurred in the past 14 years. The soldier at the front who was mortally wounded with a machine gun bullet or a fragment of shrapnel lodged in his heart was not a subject for further study, either by fluoroscopy or post mortem examination. When it is considered that in this small series 5/6 of the cases represent war injuries, what a large number of similar cases must have terminated fatally! Another and smaller group are probably alive and well with unknown foreign bodies in their hearts.

In contradistinction to heart wounds (such as stab wounds), where surgical intervention must be immediate if the patient is to be given a chance to recover, the patients who have an intracardiac foreign body either present few and mild symptoms or are quite badly shocked for a short time (as in our case) and then make a rapid recovery with no residual disorder. Another notable feature in many cases is a recurrence of pain, dyspnoea and epigastric distress days, weeks or months after the injury. This brings up the question of what constitutes a basis for surgical intervention. Should operation be immediate after the injury or be delayed for a period? In general, a careful consideration of the foregoing cases would suggest the following classification of treatment:

(1) Any sharp, slender foreign body such as a needle[1, 21, 29]; a nail[27]; a metallic pipe stem[30]; or a sharp wire[32] demands immediate operation (under fluoroscopic control) with removal of the foreign body.

(2) Any body of sufficient size to extend entirely through the cardiac wall and lie with one end in the heart cavity and the other outside of the heart muscle must be considered as a po-

TABLE NO. 1

Table No. 1 — Analysis of All Cases.

Location of F. B. in Heart	No. Cases	Deaths - all causes	Percentage Mortality	Number Operations	Deaths - operative	Cases - Non-operative	Deaths non operative	Deaths — from other causes.
Entire Heart	50	11	22	19	2	31	5	4
L. Ventricle	19	3	15.8	4	0	15	2	
in wall	16	2		3	0	13	1	1 intercurrent infection
in cavity	2	0		1	0	1	0	
transfixed	1	1		0	0	1	1	
R. Ventricle	22	6	27.3	10	1	12	2	
in wall	14	2		10	1	4	1	
in cavity	8	4		0	0	8	1	1 severity other wounds
L. Atrium	4	2	50	4	1	0	1	
in wall	3	1		3	1	0	1	
transfixed	1	1		1	0	0	0	1 suicide
R. Atrium	2	0	0	0	0	2	0	
in cavity	2	0		0	0	2	0	
Unclassified -								1
Intervent. Septum	1	0		1	0	0	0	
Cardiac wall	1	0		0	0	1	0	
Ventricular wall	1	0		0	0	1	0	

tential menace. The metallic body, acting as a plug or stopper, may give way at any time and will result in a haemopericardium. This type of injury should be operated upon as soon as the patient has recovered from the primary shock and the foreign body has been accurately localized.

(3) A missile lying free in a cardiac cavity, if not enmeshed in the trabeculae carneae or the musculi papillares, must be considered as capable of being swept on into the pulmonary artery and of causing pulmonary infarction and death. There may be a retrograde migration due to gravity, as in case 2. It is conceivable that a foreign body free in the left ventricular cavity might be swept out into the general circulation and produce the symptoms of embolism. No such case occurred in this series.

From Table 1 it may be seen that in 12 cases the foreign body was in one of the heart cavities, with a total of 4 deaths. There were no operations. Two deaths were due to the severity of other wounds; one to general gas bacillus infection; and one to migration of the foreign body to the bifurcation of the right pulmonary artery on the 4th day.

In 5 cases the foreign body arrived in the heart cavities of the right side by being swept through the venous blood channels. In 2 cases the point of entry was in the iliac vein[9, 19]; in 2 others the right hepatic[14] and inferior cava[15]; and in one the femoral vein[4].

(4) The great majority of cases will require no surgical intervention. A few cases that made an excellent immediate recovery developed severe cardiac symptoms later. Some of these were of sufficient severity to indicate operation. Most of these patients recovered with unusual relief from symptoms. In only one case (17) of this series does it seem that a non-operative procedure was responsible for death.

TABLE NO. 2

ANALYSIS OF THE CAUSE OF DEATH IN 11 FATAL CASES

1. OPERATIVE DEATHS:
   1. Sudden death on fourth day following removal of foreign body from wall of left auricle.
   2. Suppurative pericarditis and pleuritis.
2. NON-OPERATIVE DEATHS (due to wound or cardiac injury):
   3. Death on fourth day—generalized gas bacillus infection.
   4. Death on thirty-fourth day—septicaemia.
   5. Death 3 hours after injury—haemorrhage.
   6. Death on fourth day—migration of bullet into right pulmonary artery.
   7. Death on tenth day—fibrinous pleurisy and pericarditis.
3. DEATHS FROM OTHER CAUSES:
   8. Intercurrent disease.
   9. Suicide.
   10. Severity of other wounds.
   11. Injury of thighs requiring amputation.

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A CLINICAL AND PATHOLOGICAL STUDY OF CHRONIC MYOCARDITIS*

BY GREENE FITZ HUGH, M.D.†

AN analysis of the clinical and pathological records of 228 selected fatal cases, was undertaken to study the relation between the clinical syndrome and the necropsy findings in cases of cardiac failure. Cases chosen for study were those in which the heart showed at autopsy hypertrophy and dilatation, fibrosis, or infarction of the myocardium. Hearts with valvular lesions were not included. All of these cases, except 28, showed during life clinical evidences of myocardial insufficiency.

The immediate causes of death in the cases chosen are recorded in Table I. They divide into two large groups: (a) those dying as an immediate result of cardiac failure, called the cardiac group in this paper (125 patients); and (b) those dying of other immediate causes, called the non-cardiac group (103 patients). In the latter an estimate has been made of the degree of cardiac failure present prior to death and tabulated in Table I.

*From the Medical Clinic of the Peter Bent Brigham Hospital.
†Fitz Hugh—Junior Visiting Physician, Boston City Hospital. For record and address of author see "This Week's Issue", page 239.

Cardiac failure, nephritis and cerebral hemorrhage combined account for 85% of the deaths in the series. Cardiac failure alone was the cause of death in 125, or 54.8%. It was present to a moderate degree in 16 cases and was a marked element, though not the principal cause of death, in 11 additional cases of the group of 103 cases not dying as the immediate result of cardiac failure.

It is well known that from a case of chronic non-valvular heart disease the heart may show hypertrophy and dilatation alone, or in combination with fibrosis of the myocardium, arteriosclerotic thickening of the small vessels or of the coronaries, or degenerative changes of the myocardium of different grades. Sir James Mackenzie[1] mentions the absence of any recognizable correlation between the clinical picture and the pathological findings in such cases. Although it is impossible to foresee, from any symptoms or physical signs during life, just what the heart of this type will present at autopsy, the frequency of occurrence of different lesions of the heart is of interest.

TABLE I

IMMEDIATE CAUSES OF DEATH IN THE SERIES

		Amount of Cardiac Failure Present in Cases not Dying a Cardiac Death			
		None	Slight	Moderate	Marked
Cardiac Failure	125				
Nephritis	55	9	29	10	7
Bronchopneumonia	17	8	7	1	1
Cerebral Hemorrhage	14	4	6	2	2
Lobar Pneumonia	6	2	3	1	0
Cerebral Thrombosis	3	2	1	0	0
Gastro-Intestinal Hemorrhage	2	1	1	0	0
Mesenteric Thrombosis	2	0	1	1	0
Infarct of Lung	1	0	0	1	0
Hyperthyroidism	1	0	0	0	1
Obliterative Endarteritis of Lungs	1	1	0	0	0
Carcinoma of Stomach	1	1	0	0	0
Totals	228	28	48	16	11

ANATOMIC DAMAGE

Anatomic damage to the heart in 125 cases with myocardial failure and the damage seen in 103 dying of some other cause are compared in Table II. The amount of myocardial fibrosis and of thickening of small blood vessels of the heart (arteries of the 3rd division and arterioles) is based on microscopic study of one or more sections. The degree of coronary arteriosclerosis is based largely on the gross study of these vessels, although they were studied microscopically also in most cases. This method of investigation was determined by a more detailed gross and microscopic study of 14 hearts discussed later.

Analysis of the whole group of 228 hearts showed that:

8.9% had marked myocardial fibrosis.
17.7% had markedly thickened small vessels.
35.1% had marked coronary sclerosis.

In the 20 hearts with marked myocardial fibrosis, marked arteriosclerotic thickening of the small vessels was present in 45%, and in 64.7% marked coronary arteriosclerosis was found. Although markedly thickened coronary arteries and small vessels occur frequently in association with each other and with marked myocardial fibrosis, it is not unusual to find a distinct change

TABLE II

PERCENTAGE DISTRIBUTION OF MICROSCOPIC FIBROSIS OF THE MYOCARDIUM

	In all Hearts (228 cases)	In Cardiacs* (125 cases)	In Non-Cardiacs† (103 cases)
None	17.3	15.3	19.5
Slight	51.3	47.4	56.5
Moderate	22.5	24.3	20.1
Marked	8.9	13.0	3.9

PERCENTAGE DISTRIBUTION OF ARTERIOSCLEROTIC THICKENING OF THE CORONARY ARTERIES

	In all Hearts (228 cases)	In Cardiacs* (125 cases)	In Non-Cardiacs† (103 cases)
None	18.3	11.4	26.5
Slight	18.3	20.6	15.7
Moderate	28.3	21.6	36.1
Marked	35.1	46.4	21.7

PERCENTAGE DISTRIBUTION OF ARTERIOSCLEROTIC THICKENING OF THE SMALL BLOOD VESSELS

	In all Hearts (228 cases)	In Cardiacs* (125 cases)	In Non-Cardiacs† (103 cases)
None	18.6	19.0	17.4
Slight	29.6	34.0	24.3
Moderate	34.1	27.0	43.7
Marked	17.7	20.0	14.6

*Cardiacs means patients dying as the immediate result of cardiac failure.

†Non-cardiacs means patients dying of other cause than cardiac failure, but showing at autopsy lesions of the heart similar to those found in patients dying as the immediate result of cardiac failure.

in any one of the three, with only moderate or slight change in either or both of the other two.

Dividing the hearts into two groups, one made up of 125 from cardiac cases (subsequently spoken of as cardiacs or cardiac group) the other embracing 103 from cases that did not die of cardiac failure (subsequently spoken of as non-cardiacs or non-cardiac group), we can compare the frequency of the above three anatomical lesions.

The percentage of frequency of marked fibrosis of the myocardium in the non-cardiac group compared to that in the cardiac group shows a distinct preponderance in the cardiac group (13% against 3.9%). In actual figures, 20 hearts showed marked myocardial fibrosis, of which 16 were cardiacs. A less evident preponderance of cardiacs over non-cardiacs occurred in the group with marked arteriosclerotic thickening of the coronary arteries, 46.4% of the cardiacs having marked coronary thickening as against 21.7% of the non-cardiacs. Arteriosclerotic thickening of the small vessels of the heart showed a less distinct preponderance in the cardiac group than was found for either marked fibrosis of the myocardium or marked arteriosclerotic thickening of the coronary arteries. The cardiac group contained 20.0% with marked thickening, the non-cardiac group 14.6%.

In the groups with moderate, slight, or no myocardial fibrosis, no significant preponderance of cardiacs over non-cardiacs occurred. The same was true for arteriosclerosis of the coronary arteries and small vessels in amounts less than the marked degree described above.

No significance can be attached to degenerative changes of the myocardium as seen in the microscopic sections, such as fat vacuoles, hyaline droplets, absence of cross striations or swollen cross striations, for cardiac failure, nephritis, pneumonia and toxins of various kinds commonly cause these changes. One form or another of myocardial degeneration was found in 68% of the cases of cardiac failure.

In 14 hearts the coronary arteries were carefully dissected to study the changes present. The method followed was to make incisions along the course of the coronaries so as to give cross sections of these arteries at many levels. Sections were taken for microscopic study from the wall of the right and left ventricle, apex, interventricular septum, anterior papillary muscle of the left ventricle and a large coronary artery. The coronary arteries may show different grades of arteriosclerosis in different branches. The anterior descending branch showed more marked arteriosclerosis than the posterior descending in four cases of the 14 studied. The first two centimeters of the right and left circumflex branch in some cases showed more marked thickening than the rest of the coronary arteries. No hearts were found where some branches were conspicuously affected, while others showed no change.

Microscopic study of the sections taken from the six different parts of the heart showed noticeable variation in the amount of thickening of the small vessels. The same was true of the amount of degeneration of the muscle and, to a smaller degree, the amount of interstitial fibrosis of the myocardium. The evidence obtained from this more detailed work substantiates the lack of correlation between the amount of arteriosclerotic thickening of the coronary arteries and the small vessels. It shows that the thickening of the small vessels varies in different parts of the heart and that either the coronary arteries or the small vessels may be markedly thickened without causing myocardial fibrosis.

RELATION OF HYPERTROPHY TO CARDIAC DECOMPENSATION

It is common experience that cardiac decompensation appears after a certain amount of hypertrophy has occurred. Table III, with the hearts classified according to weight, shows the percentage of cardiacs and non-cardiacs in each weight group. A significant preponderance does not occur in hearts weighing less than 600

TABLE III

Weights of Hearts	Per cent. Hearts in each weight group		Number	Per cent. dying a Cardiac Death		Number	Per cent. dying of causes other than Cardiac		Number
200 Gram Group	3.0%		7	0.8%		1	6.0%		6
300 " "	63.5% 9.2%		21	56% 7.2%		9	73% 12.0%		12
400 " "	20.6%		47	20.0%		25	21.0%		22
500 " "	30.7%		70	28.0%		35	34.0%		35
600 " "	19.3%		44	20.8%		26	17.0%		18
700 " "	36.5% 10.1%		23	44% 12.0%		15	27% 8.0%		8
800 " "	4.4%		10	7.2%		9	1.0%		1
900 " " and 1000	2.7%		6	4.0%		5	1.0%		1
Totals	100%		228	100%		125	100%		103

grams. There is a very marked preponderance in the 800 and 900-1000 gram groups. Why the hearts should fail more frequently after they have reached a weight of 600 grams is not known. There is no unusual feature in the clinical history or course. It seems reasonable to suppose that heavy work would cause cardiac hypertrophy, but cardiac failure is not prominent in laborers, and studies of long distance runners have shown no hypertrophy. Christian[2] suggests that possibly the grouping of the muscle around a cavity is responsible for the inadequacy of the hypertrophied muscle. Possibly after the heart enlarges to a size of 600 grams or more, the capillaries no longer supply the hypertrophied muscle fibers with an adequate amount of nourishment (Christian). The fact that the hearts of animals may hypertrophy to a tremendous degree without the onset of myocardial failure hampers experimental work along this line.

An analysis of the hearts weighing 350 grams, or less, showed that only five patients in this group had cardiac failure clinically. A sixth had marked cardiac failure, but apparently it was secondary to hyperthyroidism. One of the 5 cases had no symptoms of chronic myocarditis, but died of coronary thrombosis, thus falling into the cardiac group.

The table shows the weight of the hearts weighing 350 grams, or less, compared to the body weight.

Weight of Heart in Grams	Body Weight in Kilos.
*250	47
250	46
330	—
340	45.2
342	57
350	51

*Hyperthyroidism.

It is significant that the body weight is small in the 5 patients on whom weights are recorded.

Hypertension was present in 2 of these patients, in both of mild degree. The eye grounds of 2 others revealed changes regarded as evidence of former hypertension.

There was no unusual fibrosis of the myocardium in these cases. The coronaries were markedly thickened in only one, and there was microscopic degeneration of the muscle only in the case of coronary thrombosis. There was nothing unusual in the clinical history of any of these cases. Except for the small body weight found in this group, there seems to be no finding that would point to a heart of small weight. Furthermore, there was nothing unusual in the pathological picture.

DURATION OF LIFE AFTER APPEARANCE OF CARDIAC SYMPTOMS

The average duration of life after the appearance of cardiac symptoms in the 125 cardiac cases was 32 months. In the 16 cardiac cases with marked myocardial fibrosis, it was 33 months, indicating that the failing heart can carry on in the presence of this change, as well as it does in its absence. Cabot[3] found the duration of life was only 9 months in his series. There was no important increase in the duration of life in the group with hypertrophied hearts over the group with smaller hearts, except in the 800 gram group, where it was 54 months. As there were only 9 cases in this group, no conclusions are possible. In the 300 gram group (9 cases) the duration was 16 months, but above the 300 gram group all were close to 31 months.

A group of 42 cases in which there had been symptoms of cardiac failure three years or more was analyzed separately to find why the heart sustained activity so long. The average duration of life in these 42 cases was 6.6 years. The percentage of these patients having high blood pressure, or former high blood pressure (64.3%), is practically the same as the percentage of all cardiacs who had, or formerly had, high blood pressure (67.2%). Of the hearts in this group 59.7% weigh 600 grams or more, whereas only 44% of the hearts of the whole group of 125 cardiacs weigh 600 grams or more. The number of hearts in each of the classes—no, slight, moderate or marked fibrosis of the myocardium—was almost the same as for the whole cardiac group.

The whole group of cardiac patients was then classified according to symptoms to see if the anatomical changes found, or the duration of life, varied in the different groups. The number of times each of the symptoms of cardiac failure occurred as the first symptom is shown by percentage in Table IV. Pratt's series[4], which includes cases with valvular disease, showed fewer patients with edema and palpitation and more with precordial pain and substernal pressure. Dyspnoea appeared coincidentally with many of the other symptoms and each was listed as a first symptom in those

TABLE IV

RELATIVE FREQUENCY OF OCCURRENCE OF THE FOLLOWING CONDITIONS AS THE FIRST SYMPTOM OF CARDIAC FAILURE

Dyspnoea	66.0%
Edema	12.1%
Palpitation	8.3%
Precordial Pain	6.4%
Fatigue	2.6%
Abdominal Pain	2.1%
Unconsciousness	1.3%
Substernal Pressure	0.6%
Tachycardia	0.6%

cases. This does not change the percentage relation.

The order of appearance of the second and third symptoms was studied and although they are not listed in the table they are referred to in one or two instances where important.

The frequency of occurrence of the different symptoms of cardiac failure in the hearts classified according to the three types of pathological lesions reveals some points of interest.

The hearts showing marked arteriosclerotic thickening of the small vessels showed dyspnoea as the first symptom in 74%, or 8% more than the whole group. Edema was the first symptom in 6.4%, or 5.7% less than the whole group. The average duration of life after the appearance of symptoms of cardiac failure in this group was 2.5 years, 0.1 year less than for the whole group of 125 cases.

Dyspnoea and edema were the first symptoms in 62% and 5%, respectively, of the cases that showed marked fibrosis of the myocardium. Precordial pain was present in 14.3%, or 7.9% more than for the whole group.

The group with marked arteriosclerotic thickening of the coronary arteries showed dyspnoea as the first symptom in 9% less, and edema in 6% more cases than did the whole group. Precordial pain was present as the first symptom in 8.9% in this group,—only 2.5% more often than in the whole group. As the first, second or third symptom it was present 7.4% more times. The average duration of life after the onset of cardiac symptoms in this group was 0.1 year more than for the whole group.

In general, the type of pathological lesion had little effect on the symptoms noted, except for edema, which occurred as the first symptom half again as often in the group with marked coronary sclerosis, but in the other two groups only half as often as the average for all cardiacs. The duration of life was practically the same in all three groups.

The group with angina pectoris showed dyspnoea as the first symptom 13% less often than did the whole group. Edema did not occur as the first symptom in any cases of this group. It was present on entrance to the hospital in 10, or 66%, of the cases. Of course, precordial pain was present in every case of this group, but was the first symptom in only 40%. It is interesting that the average duration of life after the onset of cardiac symptoms was longer in the group with angina than for the whole group by 0.7 year. Possibly the anginal pain served to decrease physical activity and lessen the cardiac load, so to speak, playing the part of prescribed rest for the cardiac patient.

The cases with precordial pain as a symptom at some time in their history showed marked arteriosclerotic thickening of the coronary arteries in 61.5% of the cases, or 15.5% more than the incidence for all cardiacs.

In the 18 cases that had nocturnal dyspnoea, marked coronary arteriosclerosis occurred only one-half as frequently as it did in the whole group of 125 cardiacs, but marked myocardial fibrosis was equally common in the two groups (i.e., in 13% of each). There was no important variation in the arteriosclerotic thickening of the small vessels between this and the whole group. Present or former hypertension occurred in 77.7% of this group and in only 67.2% of all cardiacs. The average duration of life after the onset of cardiac symptoms in this group was 3 years. They lived an average of only one year after the onset of nocturnal dyspnoea.

The group of cases that showed palpitation as the first symptom showed no essential variation in the incidence of markedly thickened coronary arteries, small vessels, marked myocardial fibrosis or hypertension, past or present, from that found for the whole group of 125 cardiac cases.

Exclusive of the group of cases with coronary thrombosis and angina pectoris, there were only two cases that entered the hospital without edema and only two that gave a story of dyspnoea for a short period. One had dyspnoea for only one week. The heart weighed 340 grams, showing moderate fibrosis with no thickening of the small blood vessels. The patient was 85 years old and probably not active, which may explain the absence of dyspnoea. In the other case there had been dyspnoea for only three weeks, which was marked on entry to the hospital. The heart weighed 490 grams. There was slight fibrosis with moderate thickening of the small vessels. The blood pressure was normal in both cases. Both the patients that had no edema had had dyspnoea for one year. Both hearts showed markedly thickened coronaries. The blood pressure was close to 160 mm. of mercury in both. While interesting that these patients had no edema, the pathological picture did not vary in any way from many who did.

Having inquired into some general aspects of heart failure and their relation to the anatomical changes found, it is of interest to consider the relation of certain special conditions occurring with heart failure with regard to the associated anatomical changes.

ELECTROCARDIOGRAMS FROM HEARTS WITH MARKED MYOCARDIAL FIBROSIS

The electrocardiographic changes in hearts with marked myocardial fibrosis seem to depend upon factors other than the amount of fibrosis present. Obviously, destruction of the conducting system will cause a change in the electrocardiogram. There may, however, be marked myocardial fibrosis with marked coronary arteriosclerosis and cardiac infarction, both recent and healed, with a normal electrocardiographic tracing, as was the case in one of these hearts. The other 16 hearts with marked myocardial fibrosis, for which we have electrocardiographic

tracings, all showed variations from normal. Two showed no variation except left ventricular preponderance. The rest showed varying changes. Premature ventricular beats were found 7 times, auricular fibrillation, 4 times; right bundle branch block and partial heart block each occurred 3 times, and a variety of other changes occurred once or twice. Although no electrocardiographic change is characteristic of myocardial fibrosis, there is some abnormality present in the majority of cases in this group.

AURICULAR FIBRILLATION

Auricular fibrillation was present in 45, or 36%, of the 125 cardiacs. It was observed in 7 cases of pneumonia, 2 of nephritis and 7 miscellaneous cases, or 16 non-cardiac cases. It was no more prominent in the cases that showed greatly hypertrophied hearts than those in which the heart was smaller. It showed no preponderance in any of the groups—i.e., those with no, slight, moderate or marked myocardial fibrosis—the percentage distribution being almost identical.

MURMURS

Murmurs were present during life in 126, or 55% of the 228 cases. They were systolic 108, systolic and diastolic 15, and diastolic 3. In the group of 125 cardiac murmurs were present in 74, or 59.2%. They were systolic 61, systolic and diastolic 12, and diastolic 1. The weight of 17.2% of the hearts was 700 grams, or more, and 30.6% of the murmurs found in the cardiac group were from cases with hearts weighing 700 grams or more. This is almost double the normal expectancy. It is to be recalled that this analysis does not include any hearts with valvular lesions. The most reasonable explanation for the presence of murmurs in many of these cases is enlargement of the valve rings, producing a relative insufficiency, the valve leaflet no longer closing the valve orifice accurately. There are other causes not well understood, for murmurs occur without cardiac enlargement with normal valves.

FREQUENCY OF HYPERTENSION

The frequency of hypertension in a group of patients with chronic non-valvular heart disease indicates that this condition plays an important rôle in the production of heart failure, or that there is some unknown condition underlying the production of both hypertension and chronic myocarditis. Christian[6] feels that with demonstrated hypertension some other unknown factor enters to determine the marked cardiac enlargement and failure, which often takes place rapidly after a long period of almost no cardiac change during a persistent hypertension. O'Hare and Walker[7] pointed out that nicking of the arteries by the veins, or variation in the calibre of the arteries, or both, are present in the eyegrounds of patients who have, or have had, a hypertension. Yater and Wagener[8] state that about 96% of patients with heart disease due to hypertension show retinal arteriosclerosis of the hypertensive type.

An analysis of the changes in the eyegrounds was made to determine in how many cases there had probably been a hypertension in the past, in addition to the ones in which we have a record of hypertension. Examination of the eyegrounds was made in 128 patients, of whom 96 were known to have hypertension, or had had it in the past. Only 8.3% of these 96 patients did not show the changes described by O'Hare and Walker as characteristic of hypertension, so that we may assume the changes are good evidence of former hypertension.

In thirteen cases of chronic non-valvular heart disease in which there was no record of a hypertension there were eyeground changes consistent with the condition. Added to the 71 cardiac cases in which we know there was a hypertension, there is a total of 84, or 67.2%, of the 125 cardiac cases in which there was a hypertension at some time. Doubtless the percentage would be higher if we had eyeground observations in all the cardiac cases. Cabot[9] says that 77% of all heart disease is due to simple hypertrophy and dilatation (or hypertensive cardiovascular disease) without valve lesions. Christian[10] finds that they comprise 61.1% of the patients with heart disease admitted to the wards of the Peter Bent Brigham Hospital.

In addition to 151 cardiac and non-cardiac patients on whom we have a record of hypertension, 20 show eyeground changes of this condition, making a total of 171. The 84 cardiac patients with a similar history of hypertension represent 49.1% of 171. Thus, although 67.2% of the 125 patients dying of cardiac failure have high blood pressure, only 49.1% of the 171 patients with hypertension died of cardiac failure. Hearts with marked myocardial fibrosis, markedly thickened small vessels, or coronary arteries, showed no significant preponderance in the hypertensive group.

The ability of the heart to withstand high blood pressure for a long period of time is shown by the following six cases. All these patients had had hypertension for 10 years or more (20 years in one case), and the blood pressure was above 200 in all but one on entry here. The hearts weighed 560 grams, or less, with the exception of one which weighed 700 grams. Only three patients complained of any dyspnoea, and edema was present on admission to the hospital in only two of these. Only two died of cardiac failure, although the cardiac element was marked in a third, who died of nephritis. Four of the six hearts showed moderately thickened, and one markedly thickened, small vessels. Three showed markedly thickened coronaries and three showed moderate fibrosis of the myocardium. It seems likely that if hypertension alone can cause

cardiac failure, all these six patients should have had it.

MYOCARDIAL INFARCTION

Thirty-one, or 24.8%, of the 125 cardiacs were cases of cardiac infarction. Precordial pain, the common symptom of this disorder, was present in only 5 cases; in a sixth there was substernal pressure. Abdominal pain was present in 4, a story of angina without recent exacerbation in 4. Pain was not a part of the story in 14 cases, almost half of the whole group.

A previous history or eyeground changes suggesting hypertension were present in 58% of the 31 patients with cardiac infarction, somewhat less than the frequency for all cardiacs. Arteriosclerotic thickening of the small vessels was in almost the same proportion in the coronary occlusion group as for all cardiacs. Moderate or marked fibrosis of the myocardium was seen in 9% more cases of coronary occlusion than was found for the whole group of 125 cardiacs. There were markedly thickened coronaries in 15.1% more cases of coronary occlusion than was found for the whole group of 125 cardiacs. Edema occurred in 42% of the cases. Thus, there was no important change in the coronary occlusion group not found in the whole group of cardiacs, except the 15.1% preponderance of marked coronary arteriosclerosis.

ANGINA PECTORIS

There were 15, or 12%, of the 125 cardiacs that had angina pectoris. The frequency of hypertension (73%) and of hearts with markedly thickened small vessels (20%) in this group with angina pectoris is practically the same as that found for all cases with cardiac decompensation in this series. Although myocardial fibrosis was absent in only one case of angina pectoris, it was present to a marked degree in only one. It was slight in 8 and moderate in 5 cases. The percentage of hearts having marked, moderate, slight or no arteriosclerotic thickening of the coronary arteries was so similar to the same percentages for the whole group of 125 cardiacs that the point is worth emphasis.

Keefer and Resnik[11] feel that angina pectoris is due to anoxemia of the myocardium, and they say that in every case of genuine angina pectoris there is an anatomical lesion that may produce anoxemia of the myocardium. They cite the difficulty of explaining the mechanism by which anoxemia calls forth a paroxysm of pain. Allbutt and others have reported cases of angina pectoris without coronary disease, which is further proof that some factor other than coronary arteriosclerosis is necessary to produce the syndrome. The pain of angina pectoris often ceases with the onset of auricular fibrillation or congestive failure. In many cases without angina pectoris just as much coronary disease and myocardial fibrosis is present as in cases with angina pectoris. In this study, although it includes only 15 cases of angina pectoris, anatomical damage is no more frequent or severe in these 15 cases than in the whole group of 125 cases. Gross[12] and Wearn[13] have studied the blood supply to the heart and both emphasize the rich anastomoses that may be found. The degree of obstruction that may be compensated for by anastomoses makes the explanation of angina pectoris on an anatomical basis difficult, if anoxemia causes the pain. Although we find anatomical damage in cases with angina pectoris, it is about as common in the cases that do not show that symptom. If anatomical damage causes angina pectoris, we do not know in what location it must be, or in what form it must occur to cause it. One patient may die of simple cardiac decompensation and another of angina pectoris, and both will show the same degree of recognizable anatomical damage to coronary arteries, small vessels and myocardium.

CONCLUSIONS

A study was made of 228 cases showing myocardial fibrosis, hypertrophy, dilatation or myocardial infarction at autopsy to determine the relation between the clinical course and the condition of the heart at autopsy. Of the 228 cases 125 died of cardiac failure (cardiac group); 55 died of nephritis; 14 of cerebral hemorrhage, and 34 of miscellaneous causes making up the non-cardiac group.

Sixteen of 20 cases with hearts showing marked myocardial fibrosis died of cardiac failure.

Thirteen per cent. of the hearts in the group with cardiac failure had marked myocardial fibrosis, 20.0% had marked arteriosclerotic thickening of the small blood vessels, and 46.4% had marked arteriosclerotic thickening of the coronary arteries. These percentages were distinctly less in the non-cardiac group. There was no preponderance of cardiacs over non-cardiacs for degrees of damage less than the marked degree described above.

There was only a rough correlation between myocardial fibrosis, arteriosclerotic thickening of the coronary arteries, and of the small vessels in the heart studied.

Heart failure is more common in the group with hearts weighing 600 grams or more.

Aside from the fact that the body weights were small, there was no unusual feature in the clinical history or autopsy findings in patients dying of myocardial failure with hearts weighing 350 grams or less.

The average duration of life after the appearance of cardiac symptoms was 32 months for the cardiac group (125 cases). It was 33 months for the group with marked myocardial fibrosis. In a group of 42 cases with an average duration of 79 months, the percentage distribution of myocardial fibrosis was practically the same as in the whole group of 125.

Dyspnoea was slightly more common and

edema slightly less so, as the first symptom where the hearts showed markedly thickened small vessels. Where the coronary arteries were markedly thickened, dyspnoea was slightly less common and edema slightly more common; precordial pain was present as the first symptom in 8.9% in this group with markedly thickened coronaries,—only 2.5% more than for all cardiac cases.

Edema was less frequent where the hearts showed marked myocardial fibrosis.

The cases with angina pectoris lived 0.7 years longer than the average duration for all cases.

The electrocardiographic changes in hearts with marked myocardial fibrosis were not characteristic.

Auricular fibrillation was present in 36% of the cardiac cases. No type of heart showed a marked frequency of involvement.

Murmurs were present in 59.2% of the cardiac cases. They were more common with an enlarged heart.

67.2% of the cardiac cases showed hypertension, past or present. The incidence of the different anatomical lesions of the heart was the same as for all cardiacs.

Cardiac infarction occurred in 24.8% of the 125 cardiac cases. Pain was absent in almost half of the cases. Coronary arteriosclerosis was marked in 15.1% more cases in this group. Marked myocardial fibrosis was slightly more common. Arteriosclerotic thickening of the small vessels was almost the same in both groups.

Angina pectoris was present in 12% of the cardiac cases. Hypertension and markedly thickened small vessels were the same in this group as for all cases. The incidence of coronary arteriosclerosis was practically the same.

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THE TREATMENT OF ESSENTIAL HYPERTENSION*

BY ROBERT STERLING PALMER, M.D.†

HEART disease causes the death of nearly two hundred thousand persons in the United States yearly, indeed is the chief cause of death and first among diseases producing invalidism. Moreover, it has been estimated that forty per cent. of heart disease is associated with arteriosclerosis and hypertension[1, 2, 3]. Any investigation, therefore, of causes, prevention, or treatment of hypertension is an attack upon a major medical problem.

One can always find a variety of opinions on what constitutes systolic hypertension. Most investigators and insurance statisticians agree that 140 or possibly 150 mm. of mercury is the upper limit of normal systolic pressure for men over 20 years of age and a limit somewhat lower than this for women[4, 5, 6, 7, 8, 9, 10]. This assumes an observation under favorable circumstances as regards nervous excitement. In later middle age this limit may be raised to 160. The normal pressure in old age tends to fall again.

Whether a prime object in the treatment of hypertension should be the lowering of the blood pressure is sometimes questioned. In the absence of any definite knowledge of the etiology, aside from age and a familial predisposition, and in view of the heart strain occasioned by continued systolic hypertension with resulting cardiac invalidism and death, this does seem to be a rational end to pursue, especially where primary kidney involvement is not present.

Reduction of weight in the markedly obese by low caloric balanced diets is occasionally attended by brilliant results. Low protein and salt-free or salt-poor diets in essential hypertension, on the whole, appear to be ineffective[11, 12, 13], though some clinicians still maintain their value[14, 15]. Langstroth[16] thinks hypertension, along with some other chronic disease, may be the result of a vitamin deficiency and that protective diets will prevent or ameliorate this condition.

In the search for some specific drug or method one may forget certain less dramatic though effective measures, as pointed out by Mosenthal[17]. The chief factor in any regimen for the hypertensive is nervous relaxation. This may involve sanatorium care, limitation of business or social activity and for the physician the medico-priestly rôle of arbiter in family difficulties. Needless to say, any benefit is derived at the expense of much time on the part of the physician and unlimited coöperation from the patient[17, 18].

However, when all general hygienic measures

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From the Medical Department of the Massachusetts General Hospital.

†Palmer—Assistant in Medicine, Massachusetts General Hospital and Harvard Medical School. For record and address of author see "This Week's Issue", page 239.

have been exhausted, there remain the patients with persistently elevated systolic and diastolic pressures, which gradually increase with time, and in whom at first there are no clear signs of renal insufficiency. It is in the treatment of such patients that one hopes to find some therapeutic agent to control the excessive blood pressure, which we believe is, in part, an over-reaction on the part of the organism.

The practitioners of physical therapy have not been inactive in this field. Marx[19] found a lasting decrease in seven of twenty-one patients treated with light baths and subjective improvement was noted in nineteen. Gutman[20] favors the use of all types of physical therapy, including light baths, vibrations, autocondensation, and colonic lavage, but especially the latter. Carbon dioxide baths are also recommended[21]. Diathermy has been used and, as is so often the case, with subjective improvement but without any appreciable change in the blood pressure[22]. Animal experiments indicate that it does not affect the blood pressure favorably[23]. Torbett[24] found that 50% of his cases were helped by warm tub baths after a two or three weeks' course. Deep x-ray therapy on patients with malignant disease does not cause a fall in blood pressure[25]. Application of radium to the basal ganglions[26] and radium chloride intravenously are apparently without effect[27]. Ultra-violet rays have been found inert by Garot[28] in regard to the blood pressures of children. Kaiser and Loebel[29] have reported the blood pressure lowered an average of 30 mm. in 15 out of 18 cases by galvanization applied in the region of the cervical ganglia.

Among miscellaneous methods recommended, Rappaport's hyperventilation by deep breathing is reported to be effective[30]. Lintz[31] has commented on the small lungs shown by x-ray in patients with essential hypertension and the experiments of Vincent and Thompson[32] lend further weight to the efficacy of hyper-respiration since this method employed for 30 seconds caused a fall in blood pressure in all but four of 41 human subjects. Wollheim and Brandt[33] caused temporary reduction in the blood pressure by injecting small amounts of water intravenously, causing changes in the blood concentration and volume. Liver extract has been the subject of favorable reports[34, 35, 36]. It may be, as Burnett[37] has suggested, that the hypotensive action results from an histamine effect, such as that observed after the intravenous use of histamine in animals[38]. Paravertebral blocking of the dorsal nerves and of the splanchnic nerves has been tried[39] but has not been found practical, though a temporary fall in blood pressure was noted.

Almost unnumbered drugs have been proposed for the treatment of high blood pressure. Bismuth subnitrate[40], mistletoe[41], atropin or novatropin alone or in various combinations with other drugs[42, 43, 44], euphyllin[45] combinations of theobromine, luminal[46], iodide and the sulphocyanate salts, garlic[47], and recently watermelon seed[48, 49, 50] have been recommended for their therapeutic efficacy. Two things are especially impressive: one, that symptoms are more affected than the blood pressure, and two, that the nervous, variable, and climacteric types of hypertension are most helped. Altnow and O'Hare[51] did not find atropin and calcium effective.

Potassium sulphocyanate in our hands, at the Massachusetts General Hospital, has appeared to be of some definitely favorable effect[52]. Others[53] have not found it so. Some toxic reactions, such as dermatitis exfoliativa, have been reported recently[54]. Gager[55] has reported continued success in 70 to 80 per cent. of his cases and states that in the majority of 700 cases from the literature the results were good. We[52] found potassium sulphocyanate worked well in about 40 per cent. of cases showing hypertension, mostly of the essential type. Fourteen further observations of this drug showed that it was effectual five times. In two of these, bromides and weight loss may well have been factors. Subjective improvement was noted four times, two of these where no hypotensive action was seen.

Pacyl, a crystalline choline derivative, has been used with apparently beneficial results abroad[56, 57], not only on increased blood pressure but on the failing blood sugar regulation and the decreased intestinal glandular secretions of elderly people. The drug has been employed in eight cases with no definite hypotensive effect noted but with subjective improvement in three patients.

The statistical studies of Alvarez and Zimmermann[58] have indicated that the pressures of sexually abnormal women are definitely higher than those of sexually normal women. Such observations, together with the fact that the incidence of hypertension in women is relatively much less before than after the menopause, have naturally suggested that some substance may be elaborated by the ovary which prevents the development of hypertension. Kylin[44] has proposed the use of sexual gland extracts in treating hypertension. This would seem to be a logical mode of attack in some cases. In the menopausal type whole ovarian residue was used five times without effect on the blood pressure, with subjective improvement in one patient. A presumably effective preparation of the female sex hormone (standardized in 25 rat units per c.c.) was given intramuscularly for five or six weeks to one patient without influence on the blood pressure but with notable subjective improvement. Only one patient has been treated with potassium iodide theocalcin and apparently improved both objectively and subjectively. This patient was suffering from angina pectoris as well. The effect of drugs exerting a dilating action on the coronary arteries is to be the subject of later studies.

DISCUSSION

The astonishing number of favorable reports on the use of a large variety of drugs and other forms of treatment in hypertension is an indication that more rigid criteria for the clinical evidence of a hypotensive effect must be met. It is noteworthy that a great many of these therapeutic agents or methods are said to be especially effective in the "vasomotor," "spastic," or "variable" types of hypertension seen at earlier ages or at the menopause in women. The spontaneous variations of the blood pressure, even in the more established cases, may be very great[59]. Familiarity with the doctor, with the procedure, encouragement by interest shown, may result in remarkable falls in the blood pressure. Dr. William B. Breed's charts showing the hypotensive action of hypodermic sterile saline in control cases, while investigating liver extract in the treatment of this disease, are an excellent example.

It is suggested, therefore, that the following criteria might be useful in the clinical estimation of the hypotensive action of any given therapeutic measure:

(I) Each observation shall consist of at least ten successive determinations of systolic and diastolic pressure at one minute intervals, the same type of instrument to be used at each observation and the patient to be in the same position.

(II) On the whole, it appears advisable that this type of investigation be carried out, when possible, on ambulatory patients, exposed to the daily nervous excitements which the unhospitalized person must meet. The question of preliminary rest may be optional, provided it be always the same.

(III) Two or more, preferably more, weekly observations shall be made before starting treatment.

(IV) A fall in the average of the ten readings at successive visits should be demonstrable after a period allowed for the full effect of the treatment and this effect should be maintained at two or more further observations. The patient should be under treatment for four to six weeks.

(V) The treatment shall then be omitted and a rise in blood pressure shown after ample time has been allowed for the effect of the procedure to have abated.

(VI) Finally, a second hypotensive effect of the treatment should again be demonstrable.

It is only by satisfying some such minimal requirements as these that an adequate clinical evaluation may be made and the results of different investigations may be compared.

In the absence of specific measures, the guiding principles in the treatment of hypertension are: (I) Reduction to a minimum of nervous strain and responsibility (II) reduction of overweight in the obese; (III) special care in the early recognition of the disease in those with a cardiovascular family history, and an attempt to control its progress.

Certain patients will respond to a well ordered hygienic regimen of limited responsibility, daily rest periods, and mild exercise. This should be instituted in all cases, with the addition of such mild sedatives for the vegetative nervous system as potassium sulphocyanate, or with the use of antispasmodic drugs such as the nitrites or theobromine compounds. Balneotherapy may be used too little. The general regimen at a large spa is, not unlikely, as helpful as the baths, but regular warm tub baths may be had by anyone and may be of much value.

CONCLUSION

The recent literature on the treatment of essential hypertension is reviewed.

Minimum criteria for the clinical evaluation of hypotensive action are suggested.

The general principles of the management of essential hypertension are discussed.

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POSTPARTUM ECLAMPSIA WITH THE PRESENTATION OF TWO CASES*

BY BENJAMIN TENNEY, JR., M.D.†

POSTPARTUM eclampsia is an acute toxemia of the puerperal woman accompanied by either coma or convulsions, or both. As Professor J. W. Williams[1] says, "The only absolutely characteristic feature of the disease is the presence of hepatic lesions". There may be other etiologic factors responsible for coma or convulsions in the puerperal woman. Hepatic lesions can only be demonstrated at autopsy; therefore, to make the diagnosis we have to rely on certain signs and symptoms which exclude other causes and leave only eclampsia as the proper deduction. In the first place, the patient should have shown some sign of toxemia during the preceding pregnancy or labor, most commonly either a rise in blood pressure or albumen in the urine. With this already existing toxemia, the sudden appearance postpartum of coma or convulsions accompanied by albumen in the urine and a high blood pressure should lead one immediately to suspect eclampsia. The systolic pressure in these cases is usually over 160 and may be well over 200; it may be however, much lower, as in the first case that I am presenting. In this instance the patient had her first onset of convulsions intrapartum, at which time her blood pressure was only 136. A catheter specimen taken at the onset of the coma or convulsions will show a small amount of urine, a high specific gravity, considerable albumen and blood together with casts. The blood chemistry, at the same time, will show little change from the normal, except perhaps a rise in the uric acid. A high non-protein nitrogen should immediately suggest the uremia of nephritis as a more probable diagnosis than eclampsia. In cerebral hemorrhage and embolism there may not be in certain instances the rise in blood pressure nor the albumen in the urine. Epilepsy has sometimes been confused with postpartum eclampsia, but while there may be a rise in blood pressure during the seizure, there is no albumen nor other signs of toxemia. A sugar test of the urine, and if possible, of the blood, will eliminate the possibility of diabetic coma.

Postpartum eclampsia is not a rare condition. Various authorities estimate its incidence as from fifteen to thirty per cent. of all eclampsia. However, the great majority of cases occur during the first forty-eight hours after birth, and cases after seventy-two hours are more rare. The etiology of postpartum eclampsia is undoubtedly the same as that of antepartum or intrapartum eclampsia, since it is the same disease. The etiology of eclampsia is still a part of the great unknown, the general consensus of opinion being that there is a toxic agent carried in the blood plasma derived from a source as yet undiscovered. Therefore, in postpartum eclampsia this poison has not been entirely eliminated at the birth of the

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†Tenney—Assistant Obstetrician, New England Baptist Hospital. For record and address of author see "This Week's Issue", page 239.

child as is the rule in preëclamptic toxemia. One would think the retention of the toxic substance after birth would cause more permanent damage and be a more fatal affair than when the poison manifests itself before labor. The contrary seems to be the case. Hingston and Mudaliar[2] published in 1927, in the *Indian Medical Gazette*, a most interesting review of a series of two hundred cases of eclampsia taken from an Indian Maternity Hospital. In this series of cases twenty-seven per cent. were antepartum, forty-seven per cent. were intrapartum and twenty-five per cent. postpartum. Their treatment of these cases was essentially conservative in that the patients were allowed to deliver normally so far as possible; morphine and other sedatives were used and veratrone was given to lower the blood pressure. The mortality figures for the series was as follows: antepartum 8.2%, intrapartum 14.56% and postpartum 3.57%. In other words, the mortality postpartum was less than half of that antepartum, and less than one-third of that intrapartum. The authors also mentioned as a clinical observation that convulsions were much less frequent postpartum.

That postpartum eclampsia is a result of preëclamptic toxemia is evidenced by the fact that the reported cases show a toxemia either before or during labor. Also it is probable that some cases reported, such as one twenty-nine days after delivery, should not be classed as true eclampsia. Ivanyi[3], in the *Zentralblatt für Gynäkologie*, reports a case of his own occurring on the fifteenth day. He also mentions eight others fourteen or more days postpartum. As to the actual time limit for postpartum eclampsia it is impossible to say, although two weeks would seem to be a fair guess.

An interesting point, inasmuch as one of my cases was a twin pregnancy, is that all eclampsia is a more frequent occurrence in multiple pregnancies. Two of the cases mentioned in current literature of postpartum eclampsia were multiple pregnancies: one twins, and one triplets. In the paper by Hingston and Mudaliar previously mentioned it was stated that the percentage of eclampsia in multiple pregnancies was four times that of single pregnancies.

In regard to the treatment of postpartum eclampsia, it is generally admitted that prompt venesection is the first and most important point. If the patient has not had a previous hemorrhage, it is safe to remove one thousand cc. of blood. In one of my two cases reported here nine hundred cc. of blood were removed. The patient's blood count was the same five days after the venesection as the day previous. There was a fall of ten per cent. in the hemoglobin. In a given case enough blood should be removed to give a drop in systolic blood pressure of at least thirty points. After the first five hundred cc. have been removed there is generally a drop in systolic pressure of at least that amount. In case of another rise in systolic pressure another five hundred cc. should be taken off immediately. This venesection should cause a cessation of the convulsions, although the coma may continue for some time. Following the venesection, the patient should be put on the Stroganoff régime, or, in other words, be deeply narcotized with sedatives. In the Stroganoff régime large quantities of morphine sulphate and chloral hydrate are used, with chloroform for the convulsions. The original medication is planned to keep the patient narcotized for twenty-four hours, but this should be repeated should she become restless or the blood pressure not remain within proper limits. Quiet and a darkened room are an essential part of the treatment. As an important feature of eclampsia is a partial or complete anuria, the kidneys must be stimulated to eliminate as much of the toxin as possible. It has been found by clinical experience that hypertonic glucose is an excellent method of producing this effect. It may be given in strength of 10-20% intravenously, 300-500 cc. This should be repeated two or three times a day until the kidneys begin to put out at least 40 ounces of fluid in twenty-four hours. As these patients are apt to vomit continuously, it is essential to force fluids intravenously, by rectum, and under the skin to prevent a rapid dehydration. As soon as the patient can take salts by mouth, it is most important that a free catharsis be started. Finally, the nursing care should be of the best. The patient is unable to take care of herself in any way, and should not be left alone for a single moment. Hallucinations and excitement are a very common part of postpartum eclampsia and may appear unexpectedly at any time. One of my patients thought that the hospital was on fire and did her utmost to get out of the window. These hallucinations may extend over some days even after the patient seems much improved, and during their convalescence peculiar mental wanderings are at times very noticeable.

There are a few points that I particularly wish to stress in regard to the two cases reported in this paper. One patient had convulsions on the sixth day and the other on the eighth day postpartum. The first patient had a previous attack of convulsions during labor, and the second had labor induced one month before term because of a toxemia of three weeks' standing. Both patients had sedatives postpartum and had free catharsis up to the onset of their convulsions. One patient did not nurse her baby. The second patient had, at the time of delivery, a massive edema of both legs. After the birth of the baby, this patient voided large amounts daily and the edema of her legs disappeared entirely before the onset of her convulsions. This same patient became blind during the eclampsia and recovered her sight entirely before discharge. Finally, the blood chemistry and renal tests on both patients showed no definite change

from the normal. At discharge each patient was albumen-free with no casts or blood cells in the urine, denoting freedom from any permanent kidney damage.

CASE A.

A married white woman, 27 years of age with a negative past history. Para IV. No complications in previous pregnancies except that her first baby was a difficult forceps delivery and was stillborn. Present pregnancy uneventful until one week before delivery when there was a rise of blood pressure to 140. One week later she was sent in to the Boston Lying-In Hospital at term with a blood pressure of 140 and a large trace of albumen. Due to toxemia she was started in labor with castor oil and quinine. Her only complaint at that time was epigastric pain. After three hours of mild labor she had two rapid convulsions. She was immediately bled of 1000 cc., the blood serum removed and the corpuscles reinjected according to Irving's technique. She was then started on the Stroganoff régime of morphine and chloral hydrate and absolute quiet. A stillborn baby was delivered normally a few hours later. The patient made a good recovery. The blood pressure for the following four days varied from 120-140; the albumen grew steadily less. On the fifth day postpartum the blood pressure was 130. Albumen, s. p. t. On the sixth day postpartum the blood pressure rose suddenly to 170, the urine became solid with albumen and the patient became irrational and developed twitching of the extremities and face. She was again bled 1000 cc. and the corpuscles reinfused. Following this she was once more put on the Stroganoff régime. The patient made a rapid recovery and in twenty-four hours the urine was albumen-free, the blood pressure 140. She was discharged ten days later. Blood pressure 110. No albumen.

Laboratory Findings—On day of delivery: N. P. N. 27. B. U. N. 13.3 Uric. Ac. 3.7 Blood Sug. 93 Chl. 442.

On day of discharge: N. P. N. 35. B. U. N. 7. U. A. 3. B. S. 80. Chl. 407.

At Discharge: No fixation of specific gravity of urine. Color test for kidney function 40%.

CASE B.

Married white woman 36 years of age. Para IV. The past history was negative except for some cardiac irregularity regarded as functional by a consultant. Three children living and well. No complications with previous pregnancies. Present pregnancy uneventful up to three weeks before delivery, when the patient developed edema of both legs. For the next three weeks the edema increased. Blood pressure 130-140. Very slight trace of albumen during the last week. A diagnosis of twins was made and confirmed by x-ray. When the patient was eight months pregnant by dates she was admitted to the hospital because the toxemia was becoming worse. Labor was induced immediately by rupture of membranes followed by ½ cc. thymophysin subcutaneousiy. The patient had a rapid labor and delivered two healthy twins three hours later; double ovum; total weight eleven pounds. Both babies did well. The patient made a good recovery from the delivery. She was put on free catharsis with salts. She passed large amounts of urine and the edema of the legs rapidly disappeared. She did not nurse her infants due to previous failure with her other children. The blood pressure stayed below 140, the urine continued to have a slight trace of albumen. On the night of the seventh day postpartum the patient complained of severe pain in the neck and was very restless. Blood pressure 150. On the following morning, the eighth day, when breakfast was brought in, the patient suddenly remarked she could not read the paper. Fifteen minutes later she had a convulsion. Blood pressure 170. Catheter specimen solid with albumen. The patient was bled immediately of 900 cc. and put on the Stroganoff régime. She had three convulsions in all. For the following twenty-four hours she was unconscious and very restless. She was given 900 cc. of 10% glucose intravenously,—300 cc. three times, four hours apart. After twelve hours of a urinary output of four ounces the patient put out forty ounces in the next twenty-four hours. For seventy-two hours after the convulsions she was extremely irrational and almost blind. Following this period all symptoms began to clear up. Five days after the convulsions the urine was albumen-free, the blood pressure 110, and the patient felt well except for an occasional attack of dizziness. At the time of discharge she was symptom-free, with normal vision.

Laboratory Findings—At time of convulsions: N. P. N. 35.

Two days later: N. P. N. 32. Blood Sug. 105. Uric. Ac. 4.4. Chl. 630.

At discharge: Urine albumen-free; sediment negative. Blood pressure 104.

At discharge: R. B. C. 4,400,000; Hb. 50%.

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REPORT OF A CASE OF PROGRESSIVE ATHETOSIS WITH LESIONS IN THE BASAL GANGLIA

BY BRONSON CROTHERS, M.D.,* AND STANLEY COBB, M.D.*

SINCE the pioneer work of the Vogts[1] and Wilson[2] a great deal has been learned about the function of the basal ganglia by clinico-pathological study of human material. Several cases of progressive athetosis have been reported recently[3, 4, 5] and the evidence is accumulating that this distressing syndrome usually occurs when there is disease of the striatum, perhaps particularly of the caudate nucleus. The etiology is still unknown, so it seems worth while to report all cases, even if the studies are

*Crothers—Assistant Professor in Pediatrics, Harvard Medical School. Cobb—Visiting Physician for Neurology at the Boston City Hospital. For records and addresses of authors see "This Week's Issue", page 259.

incomplete, in order to aid in the accumulation of evidence.

Case History: The patient was an only child, born some four weeks before term without difficulty. He was resuscitated with some trouble, but there was nothing in the history to suggest hemorrhage or other cerebral injury, except rather severe vomiting about the tenth day. He was breast fed totally for three weeks, and then supplementarily for five months under Dr. R. M. Smith's care. He was always coliky as a baby, but nothing abnormal was noted by the parents until he was 8 months old, when he did not sit up.

The child was first seen by Dr. Karlton G. Percy when he was 13 months old. At that time he was underweight, nervous, and showed athetoid movements almost choreiform in type. There was also a marked chronic intestinal indigestion. When he was about two years of age, on December 15, 1922, he was referred to Dr. Bronson Crothers. Physical examination by Dr. Crothers showed the following: Child lying in bed with universal disturbance of motion; chaotic grimacing, drooling, athetosis of hands and feet, and marked adductor spasm of legs. He could raise his head but could not sit up. His speech was fluent but almost completely unintelligible. He drooled constantly. On being placed on his feet, he assumed the scissors position. The detailed examination revealed little. He had no nystagmus or squint; the retinae were clear; no cranial nerve abnormality was made out. The limbs and trunk showed irregular stiffness with definitely increased deep reflexes. On the whole, the left side was more spastic than the right. No clonus was made out, and plantar stimulation produced athetosis rather than typical Babinski response.

The *impression* was that the child had a profound disturbance of control, motor chiefly extrapyramidal, without serious mental defect. It was assumed that the most probable cause was hemorrhage in a premature labor. A course of muscle training was advised and carried on.

At 2 years and three months of age, the child had acute otitis media.

The patient was again seen when 2½ years old. He had been having muscle training, and showed decided improvement. He reached for objects, could control his expressions better, drooled less, and made fairly satisfactory efforts to walk. After that Dr. Crothers saw him informally now and then but never examined him. However, it was clear that his intelligence was normal, and that he was continously improving.

At 3½ years of age he was seen in consultation, suffering from pneumonia with marked cerebral symptoms. The condition of the central nervous system improved slowly with muscle training.

At the age of 6 he became ill. This began with an acute rise in temperature to 103°, bronchial irritation and tracheitis and was followed by diarrhoea and a moderate amount of intestinal colic. Five days after the onset the temperature came down to 100°, and on the eighth day 99°. The right ear was opened and a small amount of serum escaped. The child was mentally active and restless. There was no stiffness of the neck, nor any other focal sign. He had no cough. In the afternoon the temperature rose to 105°. To quiet the patient small doses of hyoscine hydrobromide, with morphine, gr. 1/32 were given, and the child had a quieter night. Respiration suddenly ceased next morning, preceded by marked retraction of the neck, dilatation of the pupils and Cheyne Stokes breathing. His parents requested an autopsy and therefore the child was brought to the Pathological Laboratory, Harvard Medical School, for examination.

Autopsy (H-26-148) was performed by Dr. Henry Pinkerton at the Harvard Medical School on March 1, 1926 at 2:15 P. M., 8 hours post mortem. The findings were as follows:

Anatomic Diagnoses: Organized inflammatory process in the lungs; bilateral hydro-ureter, dilatation of the pelvis of the left kidney, undescended testicles, edema and congestion of the brain and otitis media on the right.

Microscopic Diagnoses: Chronic inflammation of the lung, with extreme fibrosis and foreign body giant cell reaction to an oily material (probably mineral oil); oil deposition in the lungs, bronchial lymph nodes and spleen (slight); chronic bronchial lymphadenitis; acute bronchiolitis and bronchopneumonia; acute splenitis; petechial hemorrhages in the mucosa of the stomach and the ileum and early focal necrosis of the liver.

(For a more detailed report see Pinkerton, Henry; *Am. Jour. of Diseases of Children* 33:259-285, Feb. 1927. Case 6.)

Brain: Removed in the usual way, by transverse scalp incision and wedge-shaped saw cut in the calvarium. The skull was normal. There was a slight increase in fluid over the cerebral cortex, but this fluid was not cloudy. There was also a very

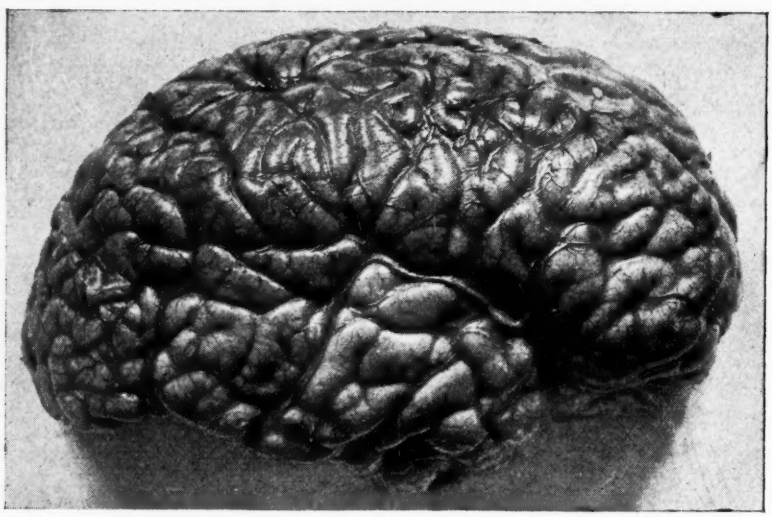

FIG. 1. Lateral View of Right Hemisphere showing somewhat unusual convolutional pattern.

slight increase in subarachnoid fluid over the entire surface of the brain, and here and there along the course of the vessels there was definite clouding of this fluid. The surface of the brain was injected, and the convolutions were definitely but not extremely flattened. The right middle ear cavity contained a small amount of greenish mucopurulent material. The left middle ear cavity was clear. The cerebral venous sinuses were congested and contained post mortem clot, but there was no evidence of thrombosis.

The brain (N-26-8) was hardened *in toto* in 10% formalin solution and was given to the Department of Neuropathology for further study.

The hardened brain, on gross examination, shows several abnormalities. The gyri are somewhat flattened and the sulci narrow, especially over the superior portions of the frontal and parietal lobes. The arrangement of the gyri is unusual: in the *right hemisphere* (fig. 1.) the Sylvian fissure is deep and long, but the operculum is not clearly defined, and it is difficult to identify the central sulcus, especially on the vertex, where the pattern of the gyri is confused and no well defined quadrate lobule can

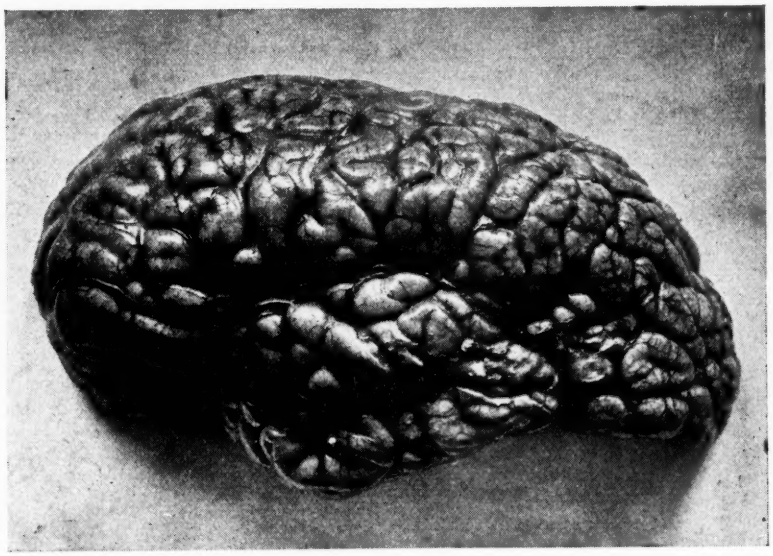

FIG. 2. Lateral View of Left Hemisphere showing abnormal convolutions especially in temporal lobe and temporo-occipital region.

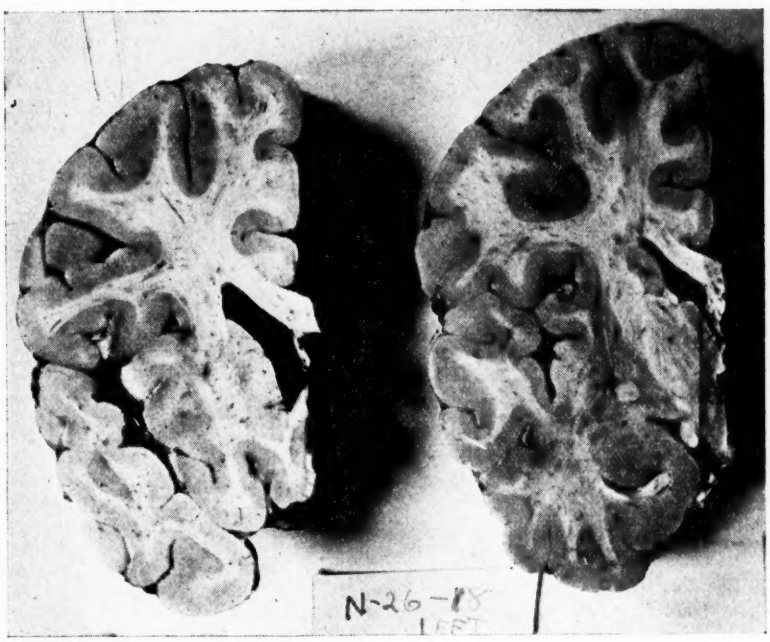

FIG. 3. Frontal Sections from Left Hemisphere showing somewhat dilated ventricle, and atrophy of caudate and lenticular nuclei. As in Fig. 4 the dark grey matter and numerous dark streaks show congestion. The gyri and sulci of the temporal lobe may be compared with the surface view in Fig. 2.

be made out. The frontal and occipital parts of the right hemisphere are not unusual in their configuration. The right temporal lobe is made up of rather large smooth gyri. This is in marked contrast to the temporal lobe of the *left hemisphere* (fig. 2) which is deeply furrowed and conspicuously divided into a small superior temporal gyrus (T_1), a large and unusually coiled middle temporal gyrus (T_2), and a laterally inconspicuous and posteriorly placed inferior temporal gyrus (T_3). The relative sizes of these gyri can be well seen also in the photograph of the sectioned brain (fig. 3). The region behind the temporal lobe where it joins the occipital lobe is unusually sunken and is composed of small irregular gyri (unfortunately somewhat injured in removing the brain). The angular and supramarginal gyri are not clearly demarcated. The gyri in the occipital lobe are smaller than usual. The central sulcus is more easily identified than on the right, but it is not well defined at its upper end where it runs into an irregular quadrate lobule. Also the precentral gyrus is unusually folded, and is separated from the frontal gyri (F_1 and F_2) by an unusually deep and straight sulcus. The inferior (orbital) aspects of both frontal lobes are deeply grooved and have very conspicuously curled gyri. Both uncinate gyri are prominent. The base of the brain, midbrain, cerebellum and medulla appear normal. There is no "pressure cone" or herniation of the cerebellum into the foramen magnum.

The hardened brain was cut into frontal sections about 15 mm. in thickness. The gross abnormalities visible are: moderate enlargement of the ventricles, and atrophy of the caudate and lenticular nuclei; the thalamus also appears somewhat small and irregular. The whole brain is congested. (See fig. 3 and fig. 4.)

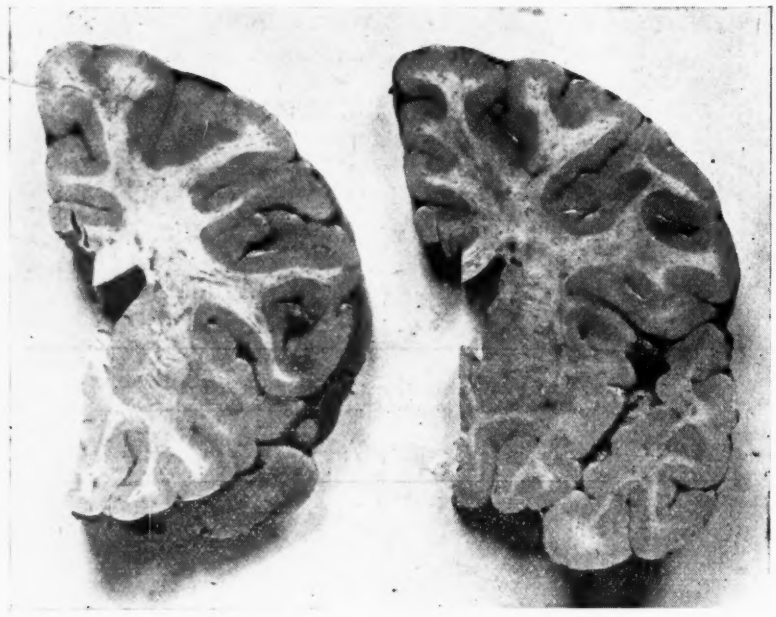

FIG. 4. Frontal Sections of Right Hemisphere showing wide ventricle, small caudate nucleus and little lenticular nucleus. The grey matter is dark because of congestion, and there are many dark spots and streaks indicating congested vessels.

MICROSCOPIC EXAMINATION

Left Hemisphere

Cerebral Cortex: There is considerable engorgement of the blood vessels; the capillaries are full of red cells. This is especially conspicuous in the white matter, but also observable in the gray matter. The white matter shows many holes, some containing purple staining mucoid material, probably due to swelling of the oligodendroglia. The nerve cells are fairly normal in appearance. There is a slight amount of satellitosis. The Ammon's horn shows pallor of the cells in Sommer's sector (H_1 of Vogt's terminology).[6]

Thalamus: There is great paucity of nerve cells, those remaining show chromatolysis. Satellites are abundant throughout, especially in close proximity to the nerve cells. There is moderate cellular gliosis. The blood vessels are very conspicuous here as in the cortex.

Lenticular Nucleus: "Whole brain" sections show marked atrophy of the lenticular nucleus. The blood vessels are numerous, and some of them have wide perivascular spaces. The nerve cells are decreased in number, and are apparently replaced by diffuse neuroglial proliferation.

Caudate Nucleus: This nucleus is also greatly atrophied. Nerve cells are scarce; gliosis is prominent; there is a great increase of blood vessels. Just beneath the ependyma there is a heavy layer of glia fibres (Fig. 5.)

Right Hemisphere

Cerebral Cortex: Blood vessels are conspicuous. Nerve cells are fairly normal. In the white matter there is the mucoid type of degeneration. In Ammon's horn the layer of pyramidal cells (pallisade layer) shows normal cells, staining clearly in the end-plate, and in Vogt's fields H_1 & H_3, in H_2 the nerve cells are less darkly stained, in H_1 (called Sommer's Sector by Spielmeyer) there are fewer cells, they are farther apart, and they all stain faintly. The cytoplasm is pale and granular and the cell bodies are elongated and the main dendrites conspicuous. The nuclei are pale; the nucleoli stand out clearly. Many "shadows" of degenerated nerve cells are seen between these pale cells.

Thalamus: Nerve cells are much fewer on this side than in the left hemisphere. There is also congestion of capillaries, moderate gliosis and satellitosis.

Lenticular Nucleus: Atrophy is very marked. There is hardly a normal nerve cell. The blood vessels are engorged, and have wide spaces about them. Gliosis is tremendous. The "ansa lenticularis" is thin and poorly myelinated.

Caudate Nucleus: Same condition of atrophy, scarcity of nerve cells, neuroglial proliferation and increase of capillaries as in left hemisphere.

COMMENT

The gross examination of the brain shows morphological anomalies of the cerebral convolutions which indicate that the brain was congenitally abnormal, but these convolutional anomalies are not rare and are not known to have any functional significance. The lesions in the basal ganglia, however, are very definite. They are best explained as processes of repair following some former injury; this process has not stopped when the original damage was healed by absorption and scar-formation, but has gone on as a progressive gliosis which has gradually involved both caudate and lenticular nuclei, and to a less extent the thalami. What the original lesion may have been is a matter of speculation. A birth injury with hemorrhage into the basal ganglia might have set up the process of repair and gliosis above described; an infantile or prenatal infection (encephalitis) might be postulated, but no evidence remains of inflammation. That the process is a developmental lack, an "abiotrophy", might be argued from the presence of the concurrent anatomical anomalies of the cerebral gyri. But it is difficult to explain the pathological picture on the basis of a congenital defect. In the present state of knowledge one must be provisionally content with the diagnosis of gliosis of unknown origin. The changes in the nerve cells and glia of the cortex are probably acute, and due to terminal toxemia, asphyxia and circulatory changes; they are not significant in relation to the principal symptomatology, the athetosis.

FIG. 5. Section through basal ganglia of Right Hemisphere. The caudate nucleus is greatly atrophied and narrow, with an irregular ventricular surface, and many blood vessels. The lenticular nucleus shows even more atrophy, and is full of large vessels. The internal capsule is fairly normal. (Weigert Stain × 3½.)

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INTERNAL HEMORRHOIDS—INJECTION TREATMENT

BY E. PARKER HAYDEN, M.D.*

FOR several years, and within the past year especially, there has appeared in medical literature an increasing number of small books and articles on the treatment of hemorrhoids by the so-called "injection method". The recent revival of interest in this form of treatment has been nearly coincident with the development of a similar method of procedure in dealing with varicose veins of the lower extremities. Injection of hemorrhoids, however, dates back many years. When first introduced it was used extensively and promiscuously, resulting in its subsequent relegation to disfavor and virtual abandonment for a period of years. My own experience with this method of treatment for the past four years, and the enthusiasm with which it is being advocated by other surgeons, suggest that a résumé of the subject at this time will not be amiss.

HISTORICAL DATA

Credit for the first use of this method is generally given to one Mitchell, of Clinton, Illinois, in 1871. The method had an inauspicious infancy, for Mitchell kept it a secret and sold the secret to a number of non-medical "pile-curers," who went about the country advertising their treatment and applying it right and left. In consequence, a number of notably poor results overshadowed probably many good results, and the medical profession reacted strongly against the method. It was condemned and forced into obscurity. That its use was extensive at first is illustrated by the fact that in 1876, five years later, Prof. E. Andrews, of Chicago, was able to collect 3300 cases.

The method was introduced in England by Swinford Edwards in 1887. It fell into disfavor there, also, but is now enthusiastically supported, as here, by reliable proctologists. Goodsall and Miles, in 1900, disposed of the entire subject in three lines, in their book, condemning the method as inadequate for cure and likely to result in abscess or fistula. Lynch, in 1914, considered the method a good one for use in limited cases, but not so satisfactory as an operation because of a higher rate of recurrence. Mummery, in 1923, discussed injection treatment at some length, considered it useful in many cases when properly employed, but not as good as operation. Hill, to whom I am indebted for instruction in the method, injects many cases but prefers the ligature operation unless contraindicated or refused. Buie approves of the method in properly selected cases, subject to careful observance of correct technique. In fact, it may be said that nearly all men with a wide experience in rectal work recognize the value of this method of treatment.

*Hayden—Assistant Surgeon to Out-Patients, Massachusetts General Hospital. For record and address of author see "This Week's Issue", page 239.

INDICATIONS AND APPLICATION

Injection treatment should be confined entirely to internal hemorrhoids. It should not be undertaken in the presence of a chronic fissure, fistula, or any perianal infection. The solutions employed are all irritating in greater or less degree, and their use produces either thrombosis or perivascular aseptic inflammation, depending on whether the solution enters a small venule or infiltrates the perivascular spaces of the pile. As external hemorrhoids produce symptoms *only* when thrombosis or inflammation exist, it is obvious that injection has no place in treatment of the external variety. Uncomplicated, internal hemorrhoids respond best to injection. Caution must be used in the treatment of internal piles which are visibly continuous with the external perianal veins. If the solution infiltrates downward under the skin, or if thrombosis extends downward, much pain and swelling will result, and it will be necessary to resort to operation for relief of these symptoms. With care this complication should not occur.

In the case of prolapsed, strangulated internal piles, one must exercise good judgment in deciding whether to inject or to operate. If the area involved is not very extensive, and if thrombosis is not already present, injection may be carried out, after which the piles must be reduced and maintained within the anal canal until their tendency to prolapse has disappeared as a result of the postinjection swelling. The patient, furthermore, must be cautioned to replace them himself if they should protrude after leaving the office. When an individual presents a large mass of prolapsed, thrombosed, internal hemorrhoids, an immediate operation is usually the best method of dealing with the situation.

INJECTION SOLUTIONS

Numerous solutions have been used in the past, among which are alcohol 96%, formalin 1-2%, perchloride of iron, ergot, and lead

acetate. None of these has survived the test of time. At present several solutions have their supporters. In England, phenol, 5 to 20% in equal parts of glycerin and water, has been used almost entirely. Quinine and urea hydrochloride, 5-10% in water, was introduced as an agent in America, in 1913, by Terrell of Richmond, and has been used with satisfaction by many others since that time. Recently Morley, in London, in the fourth impression of his book, advocates the use of 5% phenol in almond oil, injected submucously. Goldbacher, of Philadelphia, strongly urges the use of 5% phenol in Wesson or cottonseed oil. He has discarded all other solutions. These are all vegetable oils and their virtue is said to lie in the fact that they remain unabsorbed in the tissues longer than does an animal oil, but not so long as is the case with a mineral oil such as paraffin. The tendency among most writers is to get away from the stronger solutions, which not infrequently produce slough, to the weaker solutions, which accomplish their work by instituting a milder chemical reaction with resultant perivascular fibrosis and contraction. Quinine and urethane solution, as used in the treatment of varicosities of the legs, produces necrosis in hemorrhoids sufficiently frequently to render its use objectionable. Morley, until recently, has used 20% phenol solution, and his statistics quoted in this article are based on treatment with that solution. Sloughs would have been much more frequent had his technique been less painstaking. In my own experience, 5-10% quinine and urea hydrochloride in water has been very satisfactory, and the technique hereinafter described is based on injection with that agent.

TECHNIQUE

No preliminary preparation is necessary except that the lower rectum should be clear of feces. Treatment is carried out easily with the patient in right or left Sims position and the buttocks well out over the table edge. An Otis speculum is excellent, though some prefer one of the Brinkerhoff or of the Kelly type. A long, narrow, 1 c.c. glass syringe, with ordinary hypodermic needle is quite as good as are the more complicated syringes. It can be easily introduced through an Otis speculum without obscuring the operator's view. If a larger syringe is used, a long needle is necessary. Special needles can be obtained with guards to prevent the point from going too deeply. After a survey of the number and location of the hemorrhoids needing treatment, it is best to inject the smallest first in order not to obstruct view of the others. It is generally agreed that there is no contraindication to injecting several hemorrhoids at one time, as a rule, but it is probably better not to inject more than two the first time. Occasionally a sensitiveness to quinine exists, and the reaction, though not serious, is unpleasant. If not over 1 c.c. of solution is used at the first visit, a possible reaction will not be severe, and subsequent injections can be carried out with one of the other drugs. This sensitiveness is not common and I have personally never encountered it. The area to be injected is swabbed clean with a moist cotton applicator and a little antiseptic solution applied. The needle should be inserted near the upper limit of the pile and into the venous plexus but not too deeply. From ½ to 1 c.c. of solution is adequate, or enough to distend and very slightly blanch the pile. It is injected slowly and the needle left in situ for from 1 to 3 minutes before withdrawal. The swelling produced then checks bleeding from the site of puncture. When using 20% carbolic only a few drops are necessary. Injection too superficially, or in too great quantity may produce necrosis of the mucous membrane. The prick of the needle at this level is ordinarily not felt, though sometimes a little discomfort is complained of from distention of the pile. Frequently the patient has no sensation whatever, at the time, but it is not uncommon for a mild dull ache to develop in an hour or so and last about two hours.

RESULTS

One injection is usually enough to stop both bleeding and protrusion of a hemorrhoid. Since these are the most common symptoms complained of, it is necessary to explain to an individual that cessation of these symptoms does not mean that he is permanently cured. It requires usually from 3 to 8 injections in a hemorrhoid of any size to produce fibrosis sufficient for a permanent cure. Injections in a particular pile should be given not oftener than once a week. The treatment is entirely ambulatory. Patients can resume their daily activities directly following treatment, though it is better to plan the injections at a time when they can take it easy for a few hours afterward.

COMPLICATIONS

One of the strongest arguments for injection of internal hemorrhoids is the infrequency of any complications, and almost complete absence of serious ones. Morley gives a table, based on over 3000 cases, which is here quoted:

Severe pain	0.5 per cent.
Secondary hemorrhage	0.25 per cent.
Sloughing of mucous membrane (never at all severe)	about 2.0 per cent.
Perianal abscess	1 case
Irritation of the bladder	about 1.0 per cent.

Strict adherence to simple rules of technique will practically eliminate even these few complications. Morley's table does not include the occasional thrombosis which may extend to the external perianal veins, producing swelling and great pain. I have seen this happen only twice, both times in the hospital clinic. In each case

it seemed best to operate to relieve the pain, though the condition would eventually have subsided and the end result have been good without operation. It is probable that injection too low and too freely was responsible for these complications. Embolism is practically unknown.

INJECTION VERSUS OPERATION

It is not the purpose of this paper to condemn the two operative procedures now in common use in the treatment of internal hemorrhoids. A properly performed clamp and cautery or ligature operation yields a good result in a large majority of cases. The percentage of recurrences is probably less than after injection treatment. But injections can always be repeated if necessary, and thorough treatment at first usually makes a repetition of the series unnecessary. Furthermore, the method possesses advantages which cannot be denied:

1. No hospitalization is required.
2. No loss of time from business is necessary.
3. It is essentially painless.
4. No strictures result.
5. Postoperative hemorrhage is rare.
6. Postoperative catheterization is unknown.
7. Skin tabs do not result.
8. There is no mortality.

Although a hemorrhoid operation is considered among surgeons to be a minor procedure, it is not without a certain mortality. Sir Gordon Watson's figures, based on a series of 560 cases, show two deaths, each from bronchopneumonia. He gives the following statistics:

Type of operation	
Ligature	536
Clamp and cautery	15
Whitehead's operation	9
Total	560
Complications	
Deaths (bronchopneumonia)	2
Constrictions (however slight)	58
Tags	53
Fistula	6
Secondary hemorrhage	5
Submucous abscess	2
Fissure	1

Of these operative complications, constrictions can be almost entirely avoided by the skillful operator. Skin tags, on the other hand, will occur after the most carefully performed operation. The sudden disturbance of circulation when internal hemorrhoids are removed produces edema of the skin in a certain proportion of cases, and from this swelling the tags result. A very serious but fortunately a rare complication of operation may result from this sudden interruption of the circulation of the anal canal, and that is a progressive gangrene of the entire remaining mucous membrane and immediate perianal skin. The end result of this disaster, at best, is a stenosis of the anus. The only instance of this I have chanced to see was a patient at St. Mark's Hospital in London, and the surgeons there considered it rare.

IN CONCLUSION

The treatment of internal hemorrhoids by injection is a simple, safe, and satisfactory method when intelligently applied and carefully carried out. It requires no hospitalization, causes little or no discomfort to the patient, and may properly replace surgical removal in the majority of cases.

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THE RELATIVE ANTIRACHITIC ACTIVITY OF COD LIVER OIL AND IRRADIATED ERGOSTEROL (VIOSTEROL)*

BY ARTHUR D. HOLMES, M.D.,† AND MADELEINE G. PIGOTT†

THE physician confronted with the need for administering substances possessing antirachitic activity now has a variety of products at his disposal. Cod liver oil has for centuries been available as a source of antirachitic activity, although it was not known until after the discovery of vitamins that its antirachitic value was due to its high vitamin D content. In fact, it is generally agreed that cod liver oil is the richest, natural commercially available source of vitamins A and D.

Subsequent to the development of the vitamin theory, it was found that antirachitic activity may be imparted to an individual or animal by irradiating it with ultraviolet light either from natural or artificial sources. Later investigations showed that when some types of fats, oils, and food materials are irradiated, they acquire antirachitic value. From chemical studies it was found that the vitamin value of cod liver

*The Research Laboratories, the E. L. Patch Company, Boston, Mass.
†Holmes—Director of Research, The E. L. Patch Co., Stoneham. Pigott—Research Assistant, Harvard Medical School and E. L. Patch Co. For records and addresses of authors see "This Week's Issue", page 239.

and other oils resides in the unsaponifiable portion. Investigations of the unsaponifiable portion of fats and oils and of other substances possessing antirachitic value, eventually revealed that when a sterol, known as ergosterol, was prepared in pure form and subjected to ultraviolet light, it acquired antirachitic activity thousands of times greater than that of any other known substance.

With this information at hand, it appeared theoretically possible to prepare for medicinal purposes a product which would possess absolutely uniform antirachitic value. Within the past year or two, a number of products prepared in accordance with this idea have been placed on the market. In order to standardize and control such products intended for medicinal use, the Council on Pharmacy and Chemistry of the American Medical Association made a careful investigation of these new remedies and published[1] the following conclusions:

VIOSTEROL

Investigators working with various sterols discovered that ergosterol (originally derived from ergot but now obtained from other substances, mainly yeast), in crystalline form or in solution, when subjected to ultraviolet radiation develops an antirachitic (vitamin D) potency enormously greater than that of cod liver oil. For therapeutic use the ergosterol after irradiation is usually dissolved in a vegetable oil. The Council has adopted the term viosterol to designate irradiated ergosterol, and viosterol in oil to designate a preparation containing this substance dissolved in oil. The Council has also previously adopted the qualifying phrases 100 D, 5 D, etc., to designate the vitamin D potency of the various preparations as multiples of the vitamin D potency of good cod liver oil. The potency of viosterol preparations is measured by the methods used for determining the vitamin D potency of cod liver oil.

Viosterol is for use in prophylaxis and treatment of rickets and, experimentally, in other conditions arising from faulty calcium and phosphorus assimilation. It should be borne in mind that viosterol does not contain vitamin A and that harm from hypercalcemia may result from the use of too large doses of the substance.

VIOSTEROL IN OIL 100 D.—Irradiated Ergosterol in Oil.—Activated Ergosterol in Oil.—Viosterol dissolved in a vegetable oil and standardized to contain 1,333 rat units of vitamin D in each Gm., this strength being 100 times that of a potent cod liver oil used as a standard.

Actions and Uses. — See preceding article, Viosterol.

Dosage.—Daily prophylactic dose for the average infant and child, 8 to 10 drops (0.1233 to 0.1666 cc.; 2 2/3 to 3 1/3 minims); for the premature and rapidly growing infant, 15 drops (0.25 to 0.333 cc.; 5 to 7 minims); in severe cases and for adults, doses in excess of 20 drops may be given. The marketed preparations are accompanied by a standard dropper designed to deliver 3 drops to the minim.

Viosterol in oil 100 D is standardized by comparison with a potent cod liver oil containing in each 0.75 mg. 1 rat unit of vitamin D, the rat unit being defined as that amount of vitamin D which, when uniformly distributed into the standard vitamin D deficient diet—ration 2965, *Jour. Biol. Chem.* 64:263, 1925—will produce a narrow and continuous line of calcium deposits in the metaphyses of the distal ends of the radii and ulnae of standard rachitic rats.

In view of the definite details specified for the standardization of irradiated ergosterol—viosterol in oil 100 D (in the last sentence of the above), the average physician would seem to be justified in concluding: (a) that all irradiated ergosterol-viosterol 100 D—possesses the same antirachitic activity regardless of brand or manufacturer, and (b) that Viosterol possesses 100 times the antirachitic activity of average medicinal cod liver oil. However, during recent years he has learned from the reports of numerous investigators that the vitamin D content of cod liver oil is not uniform. In fact, he will immediately remember that no manufacturer of cod liver oil makes any claim that the vitamin D content of his cod liver oil is uniform. Instead, the manufacturer of cod liver oil merely guarantees a minimum vitamin D content of his cod liver oil, knowing very well that his oil will ordinarily, if not always, exceed the guaranteed minimum vitamin D content. Consequently, a physician confronted with the need for interchanging the administration of cod liver oil and Viosterol is frequently somewhat puzzled as to the relative antirachitic values of the cod liver oil which he proposes to replace by viosterol, or vice versa.

In order to obtain data in this connection, it was decided to test the antirachitic activity of a limited number of samples of irradiated ergosterol—viosterol 100 D, cod liver oil with Viosterol 5 D, and medicinal cod liver oil. Accordingly, samples of each of the three products were purchased through the drug trade. The samples were so selected that various brands of Viosterol 100 D, cod liver oil containing irradiated ergosterol 5 D, and medicinal cod liver oil were represented.

EXPERIMENTAL

The antirachitic activity of the products under investigation was determined by the method ordinarily employed in this laboratory, namely: recently weaned albino rats of comparable ancestry were fed the Steenbock diet, consisting of whole yellow corn (ground) 76%, gluten 20%, calcium carbonate 3%, and salt 1%. When the animals had been maintained on this diet for approximately three weeks, 10% of the animals, which were believed to be typical of the group, were killed and examined for evidence of rickets. The left tibia of each animal was carefully freed of tissue and placed in a 10% formaldehyde solution. Subsequently the tibia was split longitudinally and stained by treating the bones with 2% silver nitrate solution and a 5% hypo solution. When staining was complete, each bone was examined microscopically and a record was made of the extent of calcification.

Since eighty per cent. of the preliminary control animals showed pronounced rickets, it was assumed that the remainder of the group were rachitic. These animals were maintained in separate cages and their diet was supplemented by the addition of irradiated ergosterol—Vios-

terol 100 D, cod liver oil with viosterol 5 D, or medicinal cod liver oil daily for a period of eight days. The animals were killed at the end of the tenth day and the tibiae examined as indicated above.

Inasmuch as the supposed antirachitic activity of the three products, irradiated ergosterol—viosterol 100 D, cod liver oil with viosterol 5 D, the medicinal cod liver oil, was considered as being in the ratio of 100, 5, 1 respectively, the amounts of these materials fed were approximately in inverse ratio. For convenience in administrating these products, each was diluted with peanut oil. The amount of dilution was so regulated that every animal employed in the investigation received the same amount of oil as a supplement to the basal ration.

IRRADIATED ERGOSTEROL—VIOSTEROL 100 D

Six samples of irradiated ergosterol—viosterol 100 D, representing five different brands were purchased on the open market. These were biologically tested as described above. For convenience in reporting the degree of healing which occurred in the tibia of the various animals, the following abbreviations were adopted:

Negative	= no detectable indication of recalcification
+	= very slight indication of recalcification
++	= not a complete line of recalcification
+++	= narrow continuous line of recalcification
++++	= broad continuous line of recalcification
+++++	= broad continuous line of recalcification extending well into the shaft
(+)	= less than +

The results of the examination of the six samples of irradiated ergosterol—Viosterol 100 D, are reported below:

ANTIRACHITIC ACTIVITY OF IRRADIATED ERGOSTEROL—VIOSTEROL 100 D

Sample No.	Rat No.	Amount Administered mgs.	Degree of Healing
1182	812	.084	+++
	813	.084	+++
	814	.084	+++
	815	.084	+++
1218	1024	.084	++++(+)
	1025	.084	+++
	1026	.084	+++++
	1027	.084	++++
	1028	.084	+++++
1219	1014	.084	Negative
	1015	.084	Trace
	1016	.084	Trace
	1017	.084	Negative
	1018	.084	Negative
1184	796	.084	(+)
	797	.084	+++
	798	.084	+(+)
	799	.084	+++
1183	804	.084	+(+)
	805	.084	Negative
	806	.084	Negative
	807	.084	Trace
1181	788	.084	Negative
	789	.084	Negative
	790	.084	Negative
	791	.084	Negative

The results obtained for the six samples of Viosterol indicate a surprising variation in their antirachitic activity. All animals received a total of 0.672 mgs. of viosterol 100 D during the eight day experimental period. Samples Nos. 1182 and 1218 meet the specifications for antirachitic potency. Sample No. 1184 did not produce satisfactory healing as the result of administering 0.672 mgs. during the eight day feeding period. However, the specifications provide that healing shall be initiated when 0.75 mgs. of Viosterol 100 D is fed and it is possible that had 0.75 mgs. of sample No. 1184 been fed, a satisfactory degree of healing might have resulted. Sample No. 1183 gave very little healing and sample No. 1181 produced no detectable evidence of healing. If it may be assumed that the samples of irradiated ergosterol, Viosterol 100 D, under consideration, are typical of commercial Viosterol, then it may be concluded that Viosterol 100 D on the market does not possess a uniform antirachitic content.

COD LIVER OIL WITH VIOSTEROL 5 D

Three samples of cod liver oil with viosterol 5 D, representing three different brands, were tested for their antirachitic activity coincident with the tests of irradiated ergosterol—Viosterol 100 D. The results that were obtained have been tabulated below:

ANTIRACHITIC ACTIVITY OF COD LIVER OIL WITH VIOSTEROL 5 D

Sample No.	Rat No.	Amount Administered mgs.	Degree of Healing
1180	780	1.5	Trace
	781	1.5	Negative
	782	1.5	+++(+)
	783	1.5	Negative
1179	772	1.5	+++
	773	1.5	+
	774	1.5	++(+)
	775	1.5	(+)
1178	816	1.5	+++
	817	1.5	++
	818	1.5	Negative
	819	1.5	Trace

The degree of healing obtained with the three samples of cod liver oil with viosterol 5 D, which were included in this study, was not very satisfactory. With sample No. 1178 only two rats gave evidences of healing. Sample No. 1180 was even less effective, three rats out of four showing no detectable evidence of healing. Sample No. 1179, which apparently was the most potent, did not produce satisfactory healing fol-

lowing the administration of 1.5 mgs. daily. The limited data which have been obtained concerning the antirachitic activity of commercial samples of cod liver oil with viosterol 5 D seem to indicate that the antirachitic activity of these products is not uniform.

MEDICINAL COD LIVER OIL

The five samples of medicinal cod liver oil which were used in this study were quite uniform as regards physical appearance. The samples were analyzed by the methods specified in the United States Pharmacopoeia[2]. They were found to have the following chemical and physical characteristics:

Sample No.	Specific Gravity	Refractive Index	Saponification Value	Iodine Number	Free Fatty Acid Per cent.	Unsaponifiable Material Per cent.
1092	0.9227	1.4830	185.4	169.7	0.399	1.125
1089	0.9210	1.4822	186.7	163.1	0.602	0.970
1091	0.9212	1.4823	186.5	163.9	0.433	0.921
879	0.9224	1.4830	189.6	162.6	0.440	1.089
781	0.9246	1.4837	189.7	169.5	0.469	1.016

These oils were biologically tested for their antirachitic activity by the same procedure as employed for the products discussed above. The results that were obtained appear below:

ANTIRACHITIC ACTIVITY OF MEDICINAL COD LIVER OIL

Sample No.	Rat No.	Amount Administered mgs.	Degree of Healing
1092	752	8.3	+
	753	8.3	Negative
	754	8.3	++
	755	8.3	++(+)
1089	736	8.3	+(+)
	737	8.3	++
	738	8.3	+(+)
	739	8.3	++(+)
1091	744	8.3	++++
	745	8.3	+++++
	746	8.3	++++
	747	8.3	+++
879	5	6.4	+++(+)
	6	6.4	+++(+)
	7	6.4	++++
	8	6.4	+++(+)
781	58	5.0	+++++
	59	5.0	+++++
	60	5.0	+++++
	61	5.0	+++

The amount of cod liver oil, 8.3 mgs. of samples No. 1092, No. 1089, and No. 1091, fed to the experimental animals daily was approximately 100 times the amount of irradiated ergosterol—viosterol 100 D administered. In the case of samples No. 879 and No. 781 the amount administered was materially lowered with the desire to produce with these oils evidences of lack of healing. The amount administered was 6.4 and 5.0 mgs. daily, respectively. The results obtained showed that samples No. 1092 and No. 1089 did not possess sufficient vitamin D to produce satisfactory healing when 8.3 mgs. was administered for a period of eight days. The results obtained with sample No. 1091 indicated that this oil had a high vitamin D value. Inasmuch as only 6.4 mgs. daily, or a total of 51.0 mgs., of sample No. 879 and only 5.0 mgs. daily, or a total of 40.0 mgs., of sample No. 781 were ingested, these oils apparently had an unusually high vitamin D content.

COMPARISON OF ANTIRACHITIC VALUE OF DIFFERENT PRODUCTS

It is of interest to compare the antirachitic activity of those samples of irradiated ergosterol—viosterol in oil 100 D, cod liver oil with viosterol 5 D, and medicinal cod liver oil of the same brand. Accordingly, the result of the biological determination of the antirachitic activity of the various samples have been arranged in the following table in groups in accordance with their respective brands.

A COMPARISON OF THE ANTIRACHITIC ACTIVITY OF ANTIRACHITIC PRODUCTS OF THE SAME BRAND

Irradiated Ergosterol Viosterol 100 D		Cod Liver Oil Containing Irradiated Ergosterol 5 D		Medicinal Cod Liver Oil	
Sample No.	Degree of Healing	Sample No.	Degree of Healing	Sample No.	Degree of Healing
		Brand No. 1			
1184	(+)	—	—	—	—
	+++				
	+(+)				
	+++				
		Brand No. 2			
1218	++++(+)	—	—	—	—
	+++				
	+++++				
	++++				
	+++++				
1182	+++	—	—	1092	+
	+++				Negative
	+++				++
	+++				++(+)
		Brand No. 3			
1183	+(+)	1180	Trace	—	—
	Negative		Negative		
	Negative		+++(+)		
	Trace		Negative		
		Brand No. 4			
1219	Negative	1178	+++	1089	+(+)
	Trace		++		++
	Trace		Negative		+(+)
	Negative		Trace		++(+)
	Negative				
		Brand No. 5			
1181	Negative	1179	+++	1091	++++
	Negative		+		+++++
	Negative		++(+)		++++
	Negative		(+)		+++

As stated above, the amount of the three types of products administered to the experimental animals was approximately in inverse ratio to the specified antirachitic activity of these products. Accordingly, practically identical results should have been obtained for the various samples of irradiated ergosterol 100 D, and cod liver oil with viosterol 5 D. Since brand No. 1 and brand No. 2 did not contain samples of both of these products, a comparison is impossible. In the case of brand No. 3, the results obtained for sample No. 1183 and No. 1180 are very similar and both are unsatisfactory, although of the two samples, No. 1180 apparently had a slightly greater antirachitic activity than sample No. 1183. In the case of brand No. 5, negative results were obtained with all the experimental animals used in the test of sample No. 1181. Sample No. 1179 showed definite indications of antirachitic activity when fed at the level employed, but results obtained do not indicate a sufficient degree of healing. However, sufficient evidence is at hand to justify the conclusion that irradiated ergosterol, viosterol 100 D, of brand No. 5 does not possess twenty times the antirachitic activity of sample No. 1179 of cod liver oil with viosterol 5 D of the same brand. On comparing the results obtained with sample No. 1182, irradiated ergosterol—viosterol 100 D—with medicinal cod liver oil No. 1092 of the same brand, it will be noted that the results obtained with sample No. 1182 are far superior to those obtained with sample No. 1092. Thus in this instance, the sample of irradiated ergosterol, viosterol 100 D, possesses considerably more than one hundred times the antirachitic activity of medicinal cod liver oil of the same brand represented by sample No. 1092. On the other hand, comparing the results obtained with sample No. 1181 irradiated ergosterol, viosterol 100 D, brand No. 5, with those obtained for sample No. 1091, medicinal cod liver oil of the same brand, it will be noted that the results for the viosterol 100 D were in all cases negative, whereas the results with medicinal cod liver oil, sample No. 1091, were very satisfactory. Hence sample No. 1181, irradiated ergosterol—viosterol 100 D of brand No. 5, falls far short of possessing one hundred times the antirachitic activity of cod liver oil No. 1091 of the same brand.

Inasmuch as the antirachitic value of the samples of irradiated ergosterol—viosterol 100 D—varied greatly and since the same was true of the samples of medicinal oils tested, it is extremely difficult to make a comparison of the antirachitic potency of the two products. However, it is perhaps of interest to note the relative antirachitic activity of the best sample No. 1218 of irradiated ergosterol—viosterol 100 D—and the best sample No. 781 of medicinal oil. The amount of these products administered was 0.672 mgs. of sample No. 1218 irradiated ergosterol—viosterol 100 D, and 40.0 mgs. of sample No. 781 medicinal cod liver oil. In other words, instead of the irradiated ergosterol—viosterol 100 D—having one hundred times the antirachitic activity of cod liver oil, in this instance it possessed only sixty times the antirachitic activity. Furthermore, it should be noted that at this ratio the degree of healing for sample No. 781, medicinal oil, was fully equal to that of sample No. 1218, irradiated ergosterol—viosterol 100 D. From the available data, it is impossible to estimate the relative antirachitic potency of the samples of the poorer irradiated ergosterols—viosterols 100 D—with that of the samples of the better cod liver oil included in this study, but it is quite evident that the actual antirachitic activity of the poorer viosterols studied is only a small fraction of the one hundred fold which is somewhat generally assumed ratio of commercial viosterol to average medicinal cod liver oil.

These conclusions are in accord with the observation of Barnes, Brady and James[3], who call attention to the fact that "it is well known that different batches of irradiated ergosterol vary tremendously in the amount of vitamin D contained". These investigators in a clinical study of "The Comparative Value of Irradiated Ergosterol and Cod Liver Oil as a Prophylactic Antirachitic Agent When Given in Equivalent Dosage According to Rat Units of Vitamin D" found that cod liver oil (1400 rat units of vitamin D daily) prevented or cured rickets in 95% of the cases studied, whereas irradiated ergosterol (1250 rat units of vitamin D daily) prevented or cured only 44% of the cases studied. Accordingly, they concluded that "cod liver oil in a dosage of 1400 rat units of vitamin D per day was a much more satisfactory antirachitic agent than was the irradiated ergosterol in the approximately equivalent rat unit dosage. It would seem that we are not justified in considering rat units of vitamin D in irradiated ergosterol as being equivalent to the same number of rat units of vitamin D in cod liver oil as a curative or prophylactic remedy for rickets".

In a very recent clinical study of the "Comparative Value of Viosterol and Cod Liver Oil as Prophylactic Antirachitic Agents" De Sanctis and Craig[4] obtained results practically identical with those cited above. De Sanctis and Craig found that three teaspoons of cod liver oil daily (140-170 Steenbock rat units of vitamin D) prevented rickets in 97% of 100 cases studied, whereas 10 drops daily of viosterol 100 D (300 Steenbock rat units of vitamin D) prevented rickets in only 77% of 123 cases studied—23% developed clinical signs of rickets. Hence these authors conclude "Viosterol given in the dosage recommended at the present time by its various manufacturers is less effective in the prevention of rickets than cod liver oil. In this series of cases, the infants on viosterol received about twice as many rat units of vitamin D as those on cod liver oil.—From the foregoing

observations, one of two conclusions may be drawn: Either the present recommended prophylactic dose of viosterol is too small to prevent rickets or rickets is not due to a deficiency of vitamin D alone. We are inclined to believe that the second of these possibilities is true."

SUMMARY

A determination of the antirachitic activity of six samples representing five brands of irradiated ergosterol—viosterol 100 D, showed that these samples did not possess uniform antirachitic value.

It obviously follows that if various samples of irradiated ergosterol—viosterol 100 D, do not possess uniform potency, they would not have uniformly one hundred times the antirachitic value of a standard cod liver oil.

The irradiated ergosterol—viosterol 100 D—possessing the highest antirachitic activity of those tested, had less than sixty times the antirachitic activity of the best cod liver oil tested.

It is problematical as to how much the antirachitic activity of the poorer viosterols exceeded that of the better cod liver oils. However, when comparing the therapeutic value of cod liver oil and irradiated ergosterol, it is always recognized that irradiated ergosterol has only an antirachitic function whereas cod liver oil possesses this function and is rich in vitamin A as well.

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STUDY OF INFLUENZA MORTALITY IN SIX EPIDEMICS, 1920-1929

The Public Health Service has recently completed a study of influenza mortality in six epidemics. This study is based on weekly death rates from respiratory diseases recorded as influenza and pneumonia in about 95 cities of the United States during the period of 1920-1929.

From January 1, 1920, to the middle of 1929 six epidemics of more or less national extent have occurred in the United States—1920, 1922, 1923, 1926, spring of 1928, and the winter of 1928-29. Judging by the rates in the 95 cities, which seem to be only slightly higher than rates for the country as a whole, these six epidemics caused in excess of the normal or expected seasonal mortality from respiratory diseases recorded as influenza and pneumonia, approximately 250,000 deaths in the country as a whole—a total of nearly one-half as many deaths as occurred in the United States during the great pandemic of 1918-19. About one-fifth of these deaths, or 50,000, occurred during the recent epidemic of the winter of 1928-29, and two-fifths, or 100,000, occurred during the sharp epidemic of the early months of 1920.

Although these six epidemics were more or less nation-wide in extent, the various sections of the United States were by no means equally affected. In the instance of several of the smaller epidemics there appeared to be whole sections of the country that were not affected. On the other hand, certain sections of the country experienced fairly sharp epidemics in years when the rates for the country as a whole did not indicate any influenza-pneumonia deaths in excess of the median weekly rates. An example of this is an epidemic in the West South Central cities which occurred in the early months of 1925, a year when the remainder of the country appeared to be relatively free from influenza.

The point of origin and direction of progress of these six epidemics varies a great deal. Nearly every one of them arose in a different section of the country. The 1920 epidemic arose in the East North Central section and progressed very rapidly to all other sections of the country. The 1922 epidemic arose in the East, probably in the Middle Atlantic section, and the 1923 epidemic arose in the East South Central section. The 1926 and the 1928-29 epidemics arose on the West Coast, and the little epidemic of the spring of 1928 probably arose in the Mountain or West South Central section.

The rate of progress across the country varies a great deal in the different epidemics. Considering the 95 cities as a whole, however, there is more uniformity in the length of the epidemic. In 1920 the central half of the deaths occurred in a period of about 16 days. In the epidemics of 1928-29, 1922, and 1926 the central half of the deaths occurred in periods of 21, 22, and 23 days, respectively. In the small epidemic of the spring of 1928 the period was 27 days, and in the epidemic of 1923 the period was 30 days. The 1923 epidemic seems to show the least time-concentration of any of these recent outbreaks. In the case of all the other epidemics the excess rate rose and fell at about the same rate, but in 1923 the rise to the peak was considerably slower than the fall of the curve to the normal level again.—*U. S. P. H. Service.*

MEDICAL PROGRESS

PROGRESS IN THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM*

BY JAMES C. WHITE, M.D.†

SURGERY of the Sympathetic Nervous System is just beginning to come into its own and a great number of interesting papers have appeared in the last few years. As no previous report of progress has appeared in the *Journal*, I am not planning to confine this review rigidly to 1929, but to include previous work which has appeared in the past few years when it has an important bearing on the subjects under discussion.

1. PHYSIOLOGY OF THE SYMPATHETIC NERVOUS SYSTEM

The most fundamental piece of research work which has appeared in recent years is that of Cannon and his co-workers[1] on animals surviving complete excision of the cervical, thoracic, and lumbar sympathetic trunks. These animals have lived in the sheltered conditions of the laboratory in good health for many months. The animals become very sensitive to cold as they have lost the ability to conserve heat. Erection of the hairs is permanently lost, but the Horner's syndrome lasts only a short time and the peripheral arteries recover a degree of local vasomotor tone. The animals show no tendency towards vagotonia as digestion is unchanged, the heart rate is only slightly slowed, and the blood pressure remains unaltered. The basal metabolic rate is reduced about 10%. Reproduction and lactation take place in a normal manner and there are no noticeable growth changes in kittens who have had a total sympathectomy performed on one side. This shows that the sympathetic nervous system is unimportant in a protected environment, but Cannon in his previous work has shown how essential it becomes under conditions of stress and strain.

Crawford[2] has proved that the capillaries are at least partly under the influence of the vasomotor nerves. By stimulating the cervical sympathetic trunk in albino rabbits, he noticed that capillary constriction occurred in the ears following the shutdown of the arterioles by a short latent period. He showed that this was not due to secondary metabolic disturbances by paralyzing the arterioles with acetyl choline. Under these circumstances, the arterioles remained dilated while the capillaries constricted. In the presence of passive congestion, however, the capillaries may fail to contract.

Talbott, Wolff, and Cobb[3] have shown that the brain capillaries are under the control of the cervical sympathetic. They observed a 50% increase in capillary length in the corresponding cerebral hemisphere after removal of the superior cervical ganglion. They were unable to produce epileptiform attacks or to observe blanching of the cerebral cortex on stimulation of one cervical sympathetic trunk, even when the carotid on the opposite side was ligated.

Since the original cervical sympathectomies for epilepsy by Jonesco thirty years ago, little use has been made of this method of preventing attacks of cerebral angiospasm. Crile[4] has reported the use of this procedure associated with thyroid and adrenalectomy with benefit in four out of five cases.

2. PERIARTERIAL SYMPATHECTOMY

A mass of literature pro and con has appeared since Leriche modified and popularized Jaboulay's operation. The exact way in which this operation works is still a matter of uncertainty. The bulk of anatomical evidence, as summarized by Kuntz[5], is against long vasomotor fibers running down the peripheral vessels. Therefore any vasodilator effect would have to be of a reflex nature. Leriche and Fontaine[6] have brought forward evidence in favor of this view, by pointing out that stripping a section of the arterial wall produces proximal as well as distal vasodilatation and even increases circulation in the opposite extremity.

There is a certain amount of evidence that some sensory fibers do run up along the arteries. Learmonth[7] quotes a case of Foerster's in which the roots of the brachial plexus were avulsed distal to the point of attachment of the sympathetic rami. Foerster dissected out a digital nerve and artery. Stimulating the nerve produced no sensation, but when he traumatized the artery the patient felt distinct pain. Abrashonow[8] has also shown that in a dog in whom all the peripheral nerves to the leg were cut, the injection of 50% lactic acid into the femoral artery caused reactions of pain until the artery as well was divided.

Rogers and Hemingway[9] showed that a vasodilatation occurs in animals, but that it is very short lived. When periarterial sympathectomy is performed on the carotid artery of a rabbit, the vascular engorgement in the ear lasts only forty-eight hours. The division of the cervical sympathetic trunk produces a much greater and a lasting effect.

Leriche and Fontaine[10], who have performed this operation more than 300 times, feel that in

*From the Surgical Services of the Massachusetts General Hospital

†White—Associate in Surgery, Massachusetts General Hospital. For record and address of author see "This Week's Issue", page 239.

human subjects there is a definite vasodilatation which lasts up to thirty days. In many conditions this temporary increase in circulation is sufficient to produce a permanent improvement. They have published a great many most convincing cases which they have followed carefully over a period of years. On the other hand, Raess[11], Machus[12], and Muller[13] conclude that for increasing circulation in peripheral vascular disease the effects of periarterial sympathectomy are too slight and too transitory. All of these writers feel that it is of definite value in healing stubborn chronic ulcers of the leg, but here again its effects may be too short lived unless the ulcerated area has been adequately skin grafted.

Archibald[14] emphasizes one of the most valuable points in the operation's favor—its effect in relieving pain in threatened gangrene of the foot. This does not occur constantly, but in a good percentage of the cases there is definite benefit.

Sampson-Handley[15] and McClintic[16] have modified Leriche's procedure to the extent of injecting 1 to 2 c.c. of 95 per cent. alcohol beneath the adventitia of the artery instead of dissecting it off. They claim that this method is simpler and avoids the preliminary constriction of the artery, but otherwise there are no obvious differences.

3. SYMPATHETIC GANGLIONECTOMY

This operation which was first developed by Hunter and Royle[17] gives a much greater degree of vasodilatation. While it is true that the vessels in the limb regain a certain degree of local tone, yet a very considerable increase in circulation persists. Brown and Adson[18] have been able to show marked vasodilatation of the peripheral arteries three years after operation.

While Royle's lumbar operation could be counted on to interrupt the sympathetic pathways to the legs, his method of removing the stellate ganglion* through a cervical incision was not so uniformly successful. Kuntz[19] found the answer to this when he showed that the second dorsal ganglion frequently sends an important ramus to the lower cord of the brachial plexus. This cannot well be reached through a cervical approach. Henry[20] showed that both the first and second dorsal ganglia could be resected through a posterior incision, with removal of a small piece of second rib subperiosteally, and wiping the pleura away from the bodies of the two upper dorsal vertebrae. Adson[21] was the first to apply this operation.

4. EFFECTS OF SYMPATHECTOMY ON CIRCULATION

a. *Raynaud's Disease.*

Lewis[22], from a very careful set of clinical observations on the hands of patients with Raynaud's disease, concludes that the fundamental cause of the arterial spasm is not an increased tonus of the Sympathetic Nervous System, but a local process in the walls of the arteries themselves. He deduces this because local cooling of individual fingers causes a localized asphyxia, whereas a true vasoconstrictor spasm should be more widespread. The amount of constriction at a given time will depend both upon the general vasomotor tone and upon local influences. If the vasomotor element is abolished by sympathectomy, he claims that vascular spasm may still occur in the cold. In attempting to back this up from operated cases he unfortunately records only cases which have been operated through a cervical incision and therefore must have had an incomplete removal of the vasomotor nerves to the arm. While Lewis's observations are undoubtedly of great importance, operations on cases of Raynaud's disease which are being carried out by the present technic are showing consistently large increases of surface temperature and practically no instances of subsequent peripheral spasm.

Brown and Adson[18] have made a detailed study of the effects produced by cutting the sympathetic nerve supply to the extremities in cases of Raynaud's disease. They postulate:—

The abolition of sweating and the pilomotor-reaction, which extends approximately to the thighs and axillae.

The ratio of vessel lumen to wall increased from 2:1 to 3.5:1.

Histological studies show an atrophy of disuse in the muscular coat of the arteries.

Thermocouple readings show a slight vasomotor activity after operation due to local tone in the vessels.

Calorimetric studies show a greatly increased heat elimination from the sympathectomized extremities, an increase in some cases of 300%.

Leriche and Fontaine[10] believe that ganglionectomy is too radical an operation for the milder cases of Raynaud's disease. They report a large series of cases where arterial spasm has been relieved over long periods (up to five years) following periarterial sympathectomy. In the severer cases they advocate ramisectomy rather than complete removal of the sympathetic ganglia.

Our feeling at the Massachusetts General Hospital has been that the milder cases can be carried along without operation by the occasional intravenous injection of foreign protein, recommended by Allen and Smithwick[23]. In the severe cases we remove the ganglia, because we feel that anatomically it is almost impossible to be sure of a complete denervation by cutting the rami alone and because we have never been able to detect any deleterious effect on the heart from the more radical procedure.

b. *Scleroderma.*

Leriche and Fontaine[24] and Kappis[25] were able to show an arrest in the advance of this mutilat-

*The stellate or cervicodorsal ganglion is the name often applied to the inferior cervical and 1st dorsal ganglia. These two structures are either fused or separated by a short connecting trunk.

ing disease in sympathectomized extremities and a distinct improvement in favorable cases. During the past year Adson and Brown[26] and Horton, O'Leary and Adson[27] have reported five more favorable cases. They bring out the important point that 33% of patients with scleroderma have associated vasomotor spasm. These are the ones that give the best results with ganglionectomy. The advanced cases showed a cessation of vasomotor spasm with warming of the diseased parts, ulcers healed, and pain was relieved to a considerable degree. In the less marked cases the results were excellent. The skin became less tense, swelling subsided and emotional expression returned to the face. They conclude that operation should be undertaken before fibrosis and atrophy have taken place.

c. *Arthritis.*

Leriche[28] has shown that patients with traumatic arthritis often show an extraordinary recovery from a chronic state of vasomotor spasm, edema, decalcification, and pain in the injured joint after periarterial sympathectomy or ramisection.

Rowntree and Adson[29] have done dorsal and lumbar ganglionectomies in a small series of younger patients with severe periarticular arthritis. Only the following type of cases are suitable for this operation:—

1. Arthritis must be periarticular with no bone involvement except atrophy.
2. Vasomotor tone should be increased, causing cold clammy hands and feet.
3. Vasomotor tone should be decreased following foreign protein shock.
4. Patients should be under 45 and should have been given a thorough trial on medical and orthopedic measures.

All the patients which had been selected in this way showed a marked improvement following ganglionectomy.

5. EFFECTS OF SYMPATHECTOMY ON PAIN

C. H. Mayo[30] has given an interesting review of the rôle of the sympathetic nervous system in pain transmission. The vagus nerve carries no pain fibres. Langley and Gaskell believe that the pain sensations which run in the sympathetic trunks are carried by cerebrospinal fibers which go directly from the posterior root ganglia to the various viscera without synapses in the intervening sympathetic ganglia. This point is of purely academic interest and is far from being definitely settled. Another point of much more practical importance is whether some sympathetic sensory impulses do not enter the cord over anterior roots. The possibility of this route was pointed out by Learmonth[7]. It may account for the frequent failures of posterior root section to cure pain which is transmitted over the sympathetic pathways.

a. *Pain from the abdominal viscera.*

Davis, Hart and Crain[31] have investigated the pathways in the spinal cord of pain produced by forceful dilatation of the biliary ducts in dogs. All the pain could be blocked by resection of the right splanchnic nerve. In the cord, however, section of the spino-thalamic tract did not stop the pain. Nothing short of very deep section of the cord involving the destruction of a large amount of the central gray matter was successful. These experiments show why cutting the anterolateral tracts by cordotomy has often failed to control severe crises of visceral pain.

The result of this important observation centers the logical point of attack on visceral pain in the dorsal sympathetic chains. This was first brought out in the work of Laewen[32], von Gaza[33], and Leriche[34]. It has been continued by Archibald[35] and Scrimger[36], who report very encouraging results from the dorsal section of the regional sympathetic rami as they enter the intercostal nerves. A preliminary paravertebral novocaine injection in each case showed which rami it was necessary to cut.

Leriche and Fontaine[10], van Bogaert and Verbrugge[37] have reported successful results from this operation for painful gastric crises in tabes dorsalis. Every other conceivable operation including extensive section of the posterior roots, cordotomy, and cutting the vagi has been tried and has usually failed.

For pain from the pelvic viscera including idiopathic dysmenorrhoea, irritable conditions of the bladder, malignancy of the prostate, etc., Leriche and Fontaine[10], Mornard[38], and Aubert[39] report further successes following Cotte's method of resection of the presacral nerves.

b. *Pain from the thoracic viscera.*

Fewer papers have appeared during the past year advocating extensive resections of the cervical sympathetic chain for angina pectoris. This operation has carried an immediate operative mortality of up to 20% with only 60% or less of good results.

The recent physiological work of Cannon, Lewis and Britton[40] and Singer and Spiegel[41], and the anatomical studies of Jonnesco and Enarchesco[42] and Braeucker[43] show conclusively that there are important cardiac nerves below the stellate ganglion which send direct rami to the posterior cardiac plexus. None of these can be reached through an anterior incision in the neck and this undoubtedly accounts for the failures after cervical sympathectomy. As whatever impulses which traverse the cervical sympathetic trunk can only reach the spinal cord through the upper dorsal rami (there being no direct connections with the spinal cord in the neck), the point of attack in angina pectoris is shifting to the upper dorsal region. Swetlow[44] and Mixter and White[45] have interrupted the sympathetic rami to the heart by paravertebral alcohol injections in the upper dorsal region.

Summarizing the results up to the end of 1929 we have a combined series of cases giving 50% complete relief of pain, 25% more with marked improvement, and only 25% with poor results or failures. There have been no mortalities or important complications to date.

Danielopolu[46] and Leriche[34] have both warned against possible injury to the heart and lungs from interrupting the accelerator nerves which run in the upper dorsal ganglia, but Lampert[47] has removed both the stellate ganglia in dogs and then run them hard for 15 kilometers without signs of exhaustion. Furthermore, Jonnesco and Raileanu[48] have been able to find no signs of myocardial degeneration in animals after bilateral removal of the stellate ganglia. Cannon, Lewis, and Britton[10] have shown that the heart can still accelerate by the reduction of vagus inhibitor impulses and the secretion of adrenalin.

Finally, Brown and Adson[18] and numerous other clinical observers in this country have been unable to detect any sign of cardiac embarrassment after the upper dorsal ganglia on both sides have been removed.

Pain from carcinoma of the lung and pleura, also from tubercular adhesions, is transmitted over the same upper dorsal sympathetic trunks and has been successfully interrupted by Swetlow and Weingarten[19] with paravertebral injections of alcohol.

c. *Pain from the extremities.*

Nearly all the work in this field has been done by Leriche[34]. He has shown that in many cases of causalgia and amputation stump neuralgias, the pain travels over sympathetic fibers and enters the spinal cord via the communicant rami. Amputations, section of sensory nerve trunks, and even section of all the posterior roots which carry skin sensation from the extremity may fail to relieve the patient's sufferings. In a considerable number of cases in which all these methods have failed, Leriche has relieved the pain by ramisectomy.

6. EFFECTS OF SYMPATHECTOMY ON MISCELLANEOUS CONDITIONS

a. *Cervical sympathectomy in exophthalmic goitre.*

Shaw[50] believes that the excision of the middle or inferior cervical ganglia is far superior to arterial ligation in advanced cases of Graves' disease. There is an immediate recession of the exophthalmos (except in cases where the eye ball is pushed forward by retro-ocular fat), a diminution of the basal metabolic rate and tremor, and frequently a recession of the goitre. He reports two cases and quotes the series of 19 operated by Jaboulay and reviewed by Chalier[51] 6 to 14 years later. Of these three were completely cured, 9 greatly improved, 6 slightly improved and 1 a failure. Crile[52] has reported further favorable results of this method.

b. *Operations on the Vagus and the Sympathetic system in Bronchial Asthma.*

Anatomicophysiological studies indicate a bilateral control of the finer air passages mediated by both the sympathetic and the vagus nerves. Interruption of either may relax bronchial spasm.

As a result of Kümmell's work[53], stellate ganglionectomy or section of the right vagus nerve beneath its recurrent laryngeal branch have been utilized for many years by surgeons on the continent in severe cases of bronchial asthma. More than 300 such operations have been reported. Göbell[54] summarizes a series of 98 cases with a five year follow up. Neither unilateral sympathectomy nor right vagus section has given permanent results, but bilateral stellate excision has given 42% of cures plus 30% more cases improved. Leriche and Fontaine[10] have operated 5 cases of which 2 were cured and 3 failed. One of the failures was later cured by resection of his costal cartilages and cutting the right vagus nerve.

In view of the recent anatomical studies of Braeucker[43] much more favorable results should be possible if the sympathetic supply to the lungs were completely removed by operating through a posterior approach and removing the chain of upper dorsal sympathetic ganglia.

Phillips and Merle Scott[55] give a very complete summary of the literature and the result of one successful case operated by them and followed over a period of 8 months. By removing the posterior half of the right 5th rib and retracting the lung forward, they were able to cut all the important pulmonary branches of the right vagus and at the same time presumably to interrupt a majority of the sympathetic rami connecting the posterior pulmonary plexus with the upper thoracic sympathetic ganglia. This operation should give a thorough extrinsic denervation of the right lung.

c. *Effect of Vagus and Sympathetic Nerve Section on Gastro-Intestinal Activity.*

Alvarez[56] has tested the effect of resection of the vagus and splanchnic nerves in rabbits. Both sets of nerves serve as inhibitors and regulators of gastro-intestinal activity, the only sign of antagonistic action being the effect on conduction which is improved by vagotomy and injured by splanchnicotomy. He concludes that the intestinal tube possesses a large degree of autonomy and that the neuromuscular mechanism which is responsible for orderly diastalsis should be looked for in the bowel itself.

Hartzell[57] reports that dogs, in whom both vagi have been cut above the diaphragm, show a marked reduction of both free and combined hydrochloric acid. He concludes that this is because all the psychic stimulation of secretion is removed. When the vagi are cut below the diaphragm the free hydrochloric acid may rise as high as formerly, but the secretory curve is

shorter and the stomach empties one to two hours sooner.

C. H. Mayo[58], in a small series of selected cases of duodenal ulcer, has cut all the nerve branches visible when the stomach is held under downward tension without performing a gastroenterostomy. He has had good results to date and found the acids to be lowered by from 25 to 35%.

Wade and Royle[59] reported the cure of a case of Hirschsprung's disease by a left lumbar sympathectomy. Judd and Adson[60] have recently described 2 further successful cases in which persistent megacolon has been relieved by ganglionectomy. Learmonth and Markowitz[61] have shown experimentally in dogs that the thoracicolumbar sympathetic fibers running through the inferior mesenteric ganglion control the tone of the sphincter ani internus and that hyperactivity of these nerves is a cause of congenital dilatation of the colon.

d. *Effects of Sympathectomy on Spastic Conditions.*

In reading over the literature on this subject one becomes even more confused than in attempting to form an accurate estimate at the value of periarterial sympathectomy. The best that can be done is to give a short presentation of the two opposed points of view and to conclude that neither side has yet proved its point conclusively.

The bulk of physiological evidence is against a direct influence of the sympathetic nervous system in the maintenance of muscle tone. Forbes[62] has recently given a good review of the work done on muscle tone. He states that sympathetic fibers do exist in the muscles, but that there is only one type of muscle contraction which is a function of the somatic motor nerves. The apparent differences between tonus and other types of muscle contraction, as pointed out by Boeke, DeBoer, Langelaan, Hunter and Royle and Kuntz and Kerper, is superficial and not fundamental. (Much of Royle and Hunter's work was done on birds, which react very differently in this respect.) According to Forbes, there is no foundation for the view that the sympathetic nervous system controls a plastic element distinct from the contractile. The sympathetic nerve fibers, when stimulated, do not cause contraction; when cut, tone is not abolished. Cobb and Dusser de Barenne have also shown that decerebrate tonus persists when sympathetic fibers are cut. Forbes concludes that the surgical operation of ramisection for the relief of spastic paralysis rests on wholly inadequate physiologic basis.

Davis and Kanavel[63] likewise admit that there is histological evidence pointing to a dual innervation of skeletal muscle, but the experimental removal of the sympathetic trunks in cats produces no effect on normal tone. Decerebrate rigidity is unaltered by sympathectomy (with the exception of Royle and Hunter's work on goats, the literature is in agreement on this point). Carefully controlled tests before and after sympathectomy have shown no changes in paralysis agitans, degenerations of the spinal cord, cerebral hemiplegia, and Little's disease. Davis and Kanavel, as well as Forbes, believe that if sympathectomy modifies tone in any way, it is due to increased circulation and metabolism in the spastic muscles. Ranson[64] has reviewed Frank's theory that tonic impulses are carried antidromically in the posterior roots. He believes that the posterior root impulses are not important in regulating tone and that if the afferent roots of an extremity are cut it will soon regain its original tone.

Recent evidence in favor of sympathectomy for the relief of spasticity is mainly clinical.

Stewart[65] claims that in a series of twenty-seven patients rigidity, apart from contractions, has been reduced or abolished in all. The power of coördination, as well as voluntary motion, was greatly increased so that in many of his patients complex movements like walking and writing had been greatly improved.

Von Lackum[66] has operated on 40 cases and concludes that:—

1. Sympathetic ramisection offers the most effective relief in many spastic paralyses in children.

2. Optimal results can be obtained only in young patients with good mentality.

3. Tone at rest was diminished in all cases. In 12 the excessive tone on movement did not improve.

Brown and Adson[18] believe that spasticity is reduced in patients with cerebral diplegia, but that little is accomplished in cerebro-vascular disturbances. The operation is also useless following injuries to the pyramidal tracts in the spinal cord, in paralysis agitans or the athetosis of Little's disease.

Hesse[67] reports 8 cases, of which 4 with spasticity in the legs were all improved and 4 in the arm, 3 of which were benefited. He concludes that the operation has a definite value in selected cases.

To summarize, the weight of recent physiological evidence is against the direct influence of the sympathetic nervous system on the maintenance of muscle tone. It is possible that sympathectomy may produce a definite effect by improving circulation and metabolic processes in the spastic muscles. These extremities frequently show marked impairment of circulation and the patients are usually very grateful to have them lose their cold clammy feeling, even if there is no other improvement. It is therefore desirable to continue the use of the operation in selected cases until a large enough series of failures or successes have been reported to reach a universal agreement.

The writer wishes to express his thanks and appreciation to Dr. Clifford Franseen for the assistance

given him in looking up the voluminous literature on which this report is based.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

TWO CASES ILLUSTRATING THE CLINICAL FINDINGS AND MANAGEMENT OF ESSENTIAL HYPERTENSION

CARDIAC AND ANAPHYLAXIS CLINICS OF THE MEDICAL DEPARTMENT

CASE 16311

A widow of fifty-eight came to the Out-Patient Anaphylaxis Clinic October 7 complaining of asthma of twelve years' duration. Since the onset her longest period of freedom had been for two weeks. She had trouble only at night. She had no contact with animals. Her trouble was worse on excitement and overeating. Ephedrin and powders gave some relief. She did all the housework for three people, including the washing. She slept on three pillows.

Her father had asthmatic hay-fever. An uncle also had asthma.

Examination showed a rather obese, healthy looking woman. The chest showed a few scattered loud sibilant râles. Expansion was normal. The left border of cardiac dullness was 10 centimeters to the left. Midclavicular line 8. The action was regular, the rate 88. There was a faint apical systolic murmur. Blood pressure 260/150. Liver edge possibly two fingerbreadths below the costal margin. Knee jerks active. No edema of the ankles.

Intradermal tests to twelve common protein substances were substantially negative. She was given potassium iodide and stock bacterial vaccines and was referred to the Diet Kitchen for a low caloric, moderate salt and protein diet* and to the Female Medical Department for treatment of the heart condition.

X-ray examination showed the diaphragm rather high on both sides, its respiratory movements limited. The lung fields were clear. There were no mediastinal masses. The hilus shadows were prominent. There were a few calcified glands present. The heart shadow was distinctly enlarged, the right border 3.6 centimeters, left 9.4, total transverse measurement 12 centimeters, length 13.7, base 10.3, great vessels 6, chest 24.1. The increase was entirely to the left in the region of the ventricle. The curve of the ventricle was prominent. The aorta was prominent in the region of the knob. "Arteriosclerosis, hypertrophy of the left side of the heart."

In the Medical Department October 22 she reported that she had been much better in the past two weeks. She had lost five pounds, weight 145. She had no headache except perhaps a little before breakfast. She had dyspnea on going up two flights of stairs and a little questionable edema of the ankles at night. She urinated once at night. A month before the visit she had vertigo once. Examination. Pulse 120. Urine cloudy, acid, specific gravity 1.018, a trace of albumin, occasional hyalin, rare granular casts, 20 to 25 leukocytes and occasional red blood cells. Blood pressure 220/140. Nonprotein nitrogen 30 milligrams. Hinton negative.

October 28 she reported at the Anaphylaxis Clinic that she was doing well until she caught cold October 25. She was now much better, with no wheezing at all. Her blood pressure was 240/160.

November 4 she reported that she had been sick with a cold until four days ago. Now she was much better; no real asthma.

November 12 she was very much better, with not much cold and no asthma. Blood pressure 190/115. She was still taking potassium iodide.

November 18 she reported at the Anaphylaxis Clinic that she had not a bit of asthma and had never slept so well. Medical Clinic. Blood pressure 238/150, 248/165. She reported that she had been an easy bleeder. Orders: potassium sulphocyanate grains iss three times a day for one week, twice a day for one week, once a day for one week. Return in three weeks.

November 25 and December 2 she reported at the Anaphylaxis Clinic that she had had no return of asthma.

December 9 her blood pressure in the Medical Department was 230/145, 238/140, 228/125. Orders: potassium sulphocyanate grains iss daily. Return in five weeks.

December 30 she reported at the Anaphylaxis Clinic that she was wonderfully better and could sleep on one pillow.

January 20, Medical Clinic. Blood pressure 235/135, 235/125, 230/145, 230/140. Orders: potassium sulphocyanate twice a day, return in four weeks.

February 17, Anaphylaxis Clinic. In splendid condition; no asthma. She thought the reduction diet was the important thing. Medical Clinic. Blood pressure 230/160, 240/150, 240/145, 230/140, 210/140, 215/140, 230/150, 220/140, 218/140, 225/150. Orders: potassium sulphocyanate twice a day for four weeks.

March 17 she reported at the Anaphylaxis Clinic. She had had no asthma since her first

*1200 to 1300 calories. No salt to be added to food after it is cooked. No fried foods.
Breakfast. Orange or grapefruit, one serving (6 tablespoonfuls) of cooked cereal or shredded wheat, one glass of milk for cereal and coffee.
Noon meal. One serving of beef, lamb, chicken, fresh fish, egg or cheese, or one glass of milk. Two large servings of five or ten per cent vegetables. One slice of bread or three Uneeda biscuits, two level teaspoonfuls of butter, one large serving of fruit (not the sweet fruits, bananas, figs or dates).
Evening meal. Three ounces of meat or fish, two large servings of five or ten per cent vegetables, one potato, or rice or macaroni, two teaspoonfuls of butter, one cup of tea, milk for tea, one large serving of fruit.
Eight glasses of water every day.

visit. She had one slight cold three weeks before the visit, but no wheezing. At the Medical Clinic blood pressure 200/140, 214/132, 218/135, 226/140, 218/140, 218/135, 212/125, 210/135, 208/115, 198/120, 215/125. Orders: solution of potassium sulphocyanate* one teaspoonful daily for five weeks; then return.

April 7 she reported feeling very well; no headache, vertigo, tinnitus or palpitation. She was leading a quieter life and eating less. Blood pressure 209/130, 212/130, 198/135, 240/130, 205/140, 200/140, 206/140, 190/130, 200/135, 198/130.

May 12, Medical Clinic. Weight 144½ pounds. Blood pressure 252/135, 238/130, 250/135, 238/138, 246/140, 246/135, 235/140, 230/135, 218/132, 230/135. No headache, vertigo or tinnitus. She lost consciousness once after being excited.

June 9. She had now been on potassium sulphocyanate since the last visit three times a day one week, twice a day one week and then once a day. She stopped June 7. No headache, vertigo, palpitation or tinnitus. She felt less nervous since being under treatment. Otherwise there was no change from on to off medication. Blood pressure 215/130, 215/138, 210/138, 208/140, 202/120, 202/122, 198/128, 198/135, 192/130, 194/125. Orders: solution of potassium sulphocyanate one teaspoonful twice a day one week, one teaspoonful once a day next week. Return in five weeks.

CASE 16312

A married Swedish woman fifty-one years old came to the Out-Patient Medical Clinic December 6 complaining of weakness in the legs and trembling.

About three years before the visit, soon after having had all her teeth out, she had some hemorrhage from the nose. Her physician put her to bed for a month. After that she was very weak and trembling. Her catamenia ceased at that time. Her physician kept her lying down most of the time. She could walk up one flight of stairs without stopping, but was out of breath at the top. She had palpitation on exertion.

She had had an operation for "uterine trouble" and one for hernia. She had had many head colds. She had lost about fifteen pounds in the past three years. She was of a "nervous" type. Her fingers became numb when they were cold.

One sister died of heart trouble.

Examination. A well nourished woman, slightly nervous. Tonsils hypertrophied, injected. Left border of cardiac dullness one centimeter inside the midclavicular line. Apex beat inside the midclavicular line in the fifth space. Sounds loud and clear, heard all over the chest. No murmurs. Blood pressure 260/150. Abdomen, fundi and reflexes normal.

Urine examination negative.

*The stock solution of sulphocyanate contains 96 grains of the drug to eight ounces of the solvent medium thus giving one and a half grains to the teaspoonful.

X-ray examination showed the lung fields clear, the diaphragm high on both sides, sharply outlined. The aorta was a little tortuous.

Hinton negative. Non-protein nitrogen 25 milligrams.

December 23 the patient visited the Thyroid Clinic. There she gave a history of a shock three years earlier leaving her with impaired speech and gait. For some time she had felt a little warm and had disliked summer weather and also cold weather. She did not perspire and flushed very easily. She was nervous and easily irritated. She had home difficulties. Examination showed the skin warm, dry and flushed. The eyes showed slight stare, the tongue and hands coarse tremor. There was no enlargement of the thyroid. Heart sounds rapid, regular and forceful. Blood pressure 230/130. Moderate sclerosis of the vessels. Basal metabolic rate + 24. Pulse 116. Weight 63.7 kilograms.

December 23 at a second visit to the Thyroid Clinic her basal metabolic rate was + 5, pulse 96, weight 63 kilograms. She had had a cold for the past two days. It was believed that there was no thyroid disorder. She was sent back to the Medical Clinic. An additional history of very marked constipation was obtained. Her bowels moved once in three days. She still had hot flashes, though fewer than formerly. Blood pressure 215/130, 215/140, 215/150, 220/130, 210/130, 220/135. Orders: pacyl, two pills t. i. d. after meals for a week, then two pills a. m. and p. m. for a week, then one pill t. i. d. for one week.

January 13 she came in in an upset state in regard to family matters. Blood pressure 240/130, 245/140, 225/135, 205/125, 215/130, 230/160, 275/170, rest, 225/130, 215/135. Orders: pacyl, two pills a. m. and p. m. Return in three weeks.

February 3 her bowels were apparently more regular with no medication other than pacyl. She was better in all ways except that her legs were weak. Blood pressure 250/175, 245/140, 240/150, 225/140, 225/150. Orders: Russian oil and fluid extract of cascara, potassium sulphocyanate the usual course for three weeks.

February 24. Blood pressure 240/140, 225/140, 240/145, 225/140, 230/145, 230/150, 228/135, 218(?), 240/145, 218/140, 216/150. Orders: continue potassium sulphocyanate drams iss daily. Return in six weeks.

April 7. Weight 145½ pounds. She had been on potassium sulphocyanate once daily. Blood pressure 240/150, 245/150, 225/140, 232/150, 208/150, 216/150, 214/140, 210/145, 200/160. Orders: potassium sulphocyanate twice a day for four weeks. Referred to Throat Room for cautery of bleeding point in the nose if found.

May 5 she had been taking potassium sulphocyanate twice daily. No headache, vertigo or tinnitus. Palpitation when nervous. Blood pressure 250/150, 258/160, 244/150, 235/160,

230/145, 225/170, 238/150, 225/150, 235/160, 230/165.

June 9. Took digitalis grains iss daily for one week and then once daily. Complained chiefly of weakness. She was taking no potassium sulphocyanate. She had been feeling much better since a change in family arrangements. Blood pressure 200/120, 200/120, 200/120, 208/130, 194/124, 185/125, 180/140, 194/120, 194/130, 190/130. Left border of cardiac dullness 7 centimeters to the left, half a centimeter inside the midclavicular line. A slight to moderate systolic murmur at the apex. A slight systolic murmur at the base. Sounds natural. Rhythm regular. Rate 108 during examination. Orders: no medicine. Reassurance. Cascara. Return in six weeks.

Discussion

BY ROBERT STERLING PALMER, M.D., AND WALTER SWAN BURRAGE, M.D.

Case 16311

Dr. Palmer: Any middle aged or elderly patient with evidence of hypertension, arteriosclerosis and hypertrophy of the heart who complains of paroxysmal dyspnea is likely to have cardiac dyspnea or cardiac asthma rather than allergic or bronchial asthma. Mistakes are not infrequently made unless this differential diagnosis is kept in mind. This patient gives a history of paroxysmal dyspnea of twelve years' duration. We have found that in a series of two hundred and fifty cases the average duration of cardiac asthma in a hundred and seventy fatal cases was one year and three months, while the average duration in the non-fatal cases was one year and eight months[1]. The duration of this patient's attacks points towards a diagnosis of allergic asthma, as does the relief afforded by ephedrine and powders. The absence of any other signs of failure aside from some dyspnea on stairs is against cardiac asthma. The finding of scattered musical or sibilant râles through the chest is in favor of bronchial asthma. The subsequent favorable effect of stock vaccines confirms this impression.

An interesting conjecture has been advanced by Lintz[2], namely that hypertension or spasm of the minute vessels may be an allergic phenomenon. The association of asthma and hypertension is pointed out. The bacterial growth filtrates of certain organisms moreover have been shown to induce an allergic response[3]. Indeed, there are those[4, 5] who feel that nephritis may be in the nature of such a reaction. Presumably if this is true, hypertension may be an example of a more diffuse response throughout the vascular tree in comparison with the localized kidney response in nephritis. This is still entirely theoretical, and one must reserve judgment. It is necessary only to comment on the association of bronchial asthma and hypertension in this case.

The heart was enlarged on physical examination and by X-ray. Albumin and casts were found on examination of the urine. It is clear that one is dealing with a well advanced process of considerable duration.

Whether or not one should lower the blood pressure when possible may be questioned. If we believe that continued hypertension results in left ventricular strain and leads to congestive failure, then we should reduce this strain provided no untoward symptoms are produced. In this case the use of potassium sulphocyanate for twenty weeks in the dosage indicated on the accompanying chart (Chart I) produced no ill effects and exerted a definite hypotensive effect, as demonstrated by the rise in blood pressure on omitting the drug and the subsequent fall on resuming it.

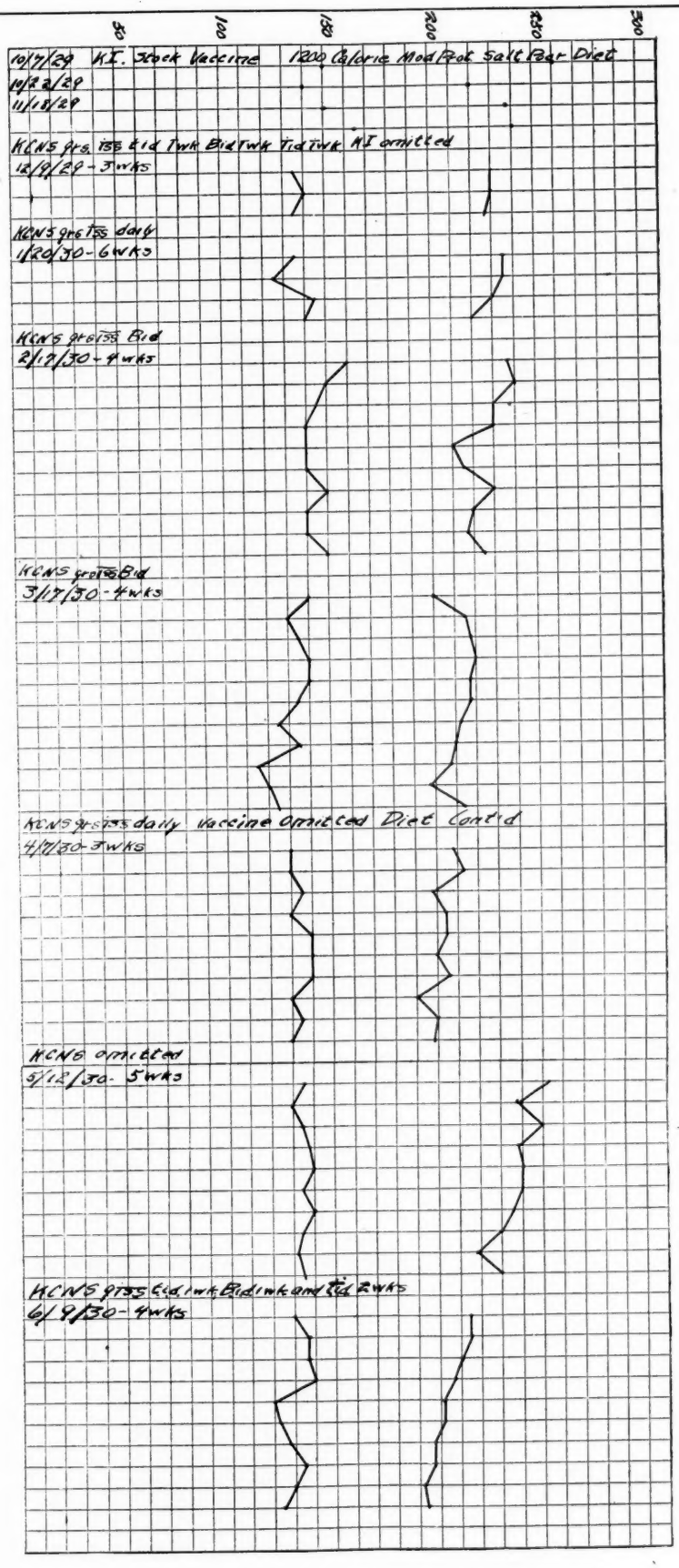
Dr. Burrage: From an allergic standpoint Case 16311 has several interesting aspects.

The presence of hypertension is an uncommon finding in uncomplicated bronchial asthma, although by no means a rare one. Hypotension is the usual observation. In this case we find prolonged, almost constant nocturnal asthma in a woman with a positive family history but with essentially negative skin tests and no clue in the past to suggest possible extrinsic factors in etiology. Arteriosclerosis, left sided cardiac hypertrophy, hypertension and renal damage are doubtless of primary importance. The low grade intrinsic type of asthma seen here is probably secondary to infections and to lowered resistance, and is based upon a fundamental inherited allergic background.

It is always difficult to evaluate the beneficial effects of treatment. Potassium sulphocyanate, potassium iodide, vaccines, weight reduction, diet and rest may all be given credit. None of these is in itself a specific against asthma, but each is doubtless a factor in raising the patient's resistance to that level at which she no longer wheezes.

Case 16312

Dr. Palmer: This second patient offers an instructive contrast to the first one. Here there is no cardiac enlargement or renal change, yet the hypertension is quite marked. The accompanying chart (Chart II) of the blood pressure determinations at each visit indicates how variable the blood pressure is, especially under emotional stress, and emphasizes the caution which must be exercised in deciding on the therapeutic efficacy of any drug. The emotional nature of this patient is brought out by the basal metabolic rate of plus 24 with a subsequent fall to plus 5. Indeed, increased metabolic rates so often found in essential hypertension may be of this nature. The trend of the chart shows clearly that neither pacyl, a choline derivative,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10/7/29 KI. Stock Vaccine 1200 Calorie Mod Prot Salt Poor Diet
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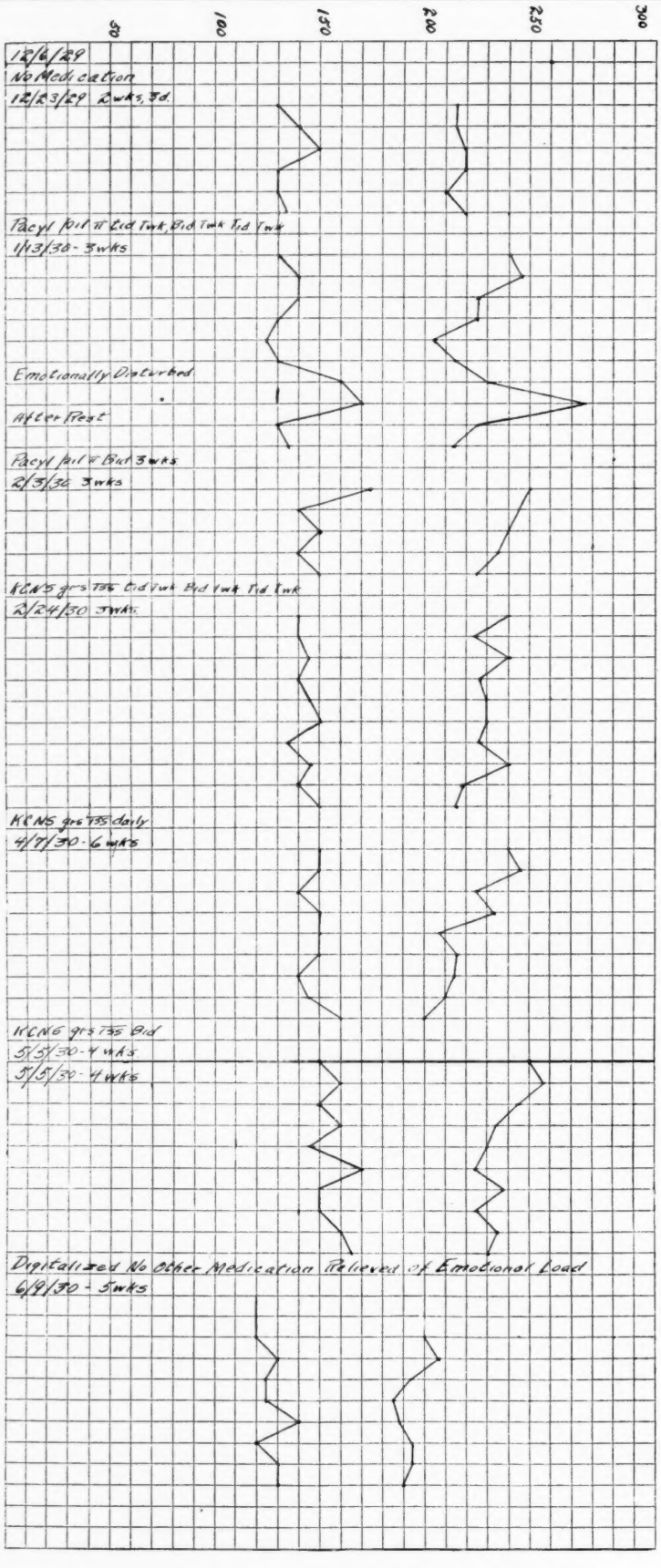
KCNS gr 6 tss t.i.d 1 wk B.i.d 1 wk T.i.d 1 wk KI omitted
12/9/29 - 3 wks
KCNS gr 6 tss daily
1/20/30 - 6 wks
KCNS gr 6 tss B.i.d
2/17/30 - 4 wks
KCNS gr 6 tss B.i.d
3/17/30 - 4 wks
KCNS gr 6 tss daily Vaccine omitted Diet Cont'd
4/7/30 - 3 wks
KCNS omitted
5/12/30 - 5 wks
KCNS gr 6 tss t.i.d 1 wk B.i.d 1 wk and t.i.d 2 wks
6/9/30 - 4 wks

CASE 16311. Chart I. The chart illustrates the variable nature of hypertension and shows that one can draw conclusions only from successive determinations and repeated observations. In this case the trend is clearly downward under potassium sulphocyanate. Omission of the drug produced a definite rise, while resuming it caused a fall in the blood pressure.



CASE 16312. Chart II. The chart shows the failure of two drugs to affect the blood pressure, while emotional stimulation resulted in a marked temporary rise, and relief of emotional strain resulted in a marked fall in the blood pressure.

nor potassium sulphocyanate had any hypotensive effect, whereas emotional disturbance at the second visit caused a sharp rise, and relief of emotional strain previous to the last visit caused a marked fall in the blood pressure.

This patient clearly has the menopausal type of hypertension, characteristically quite variable, sometimes called spastic or emotional hypertension. The absence of renal changes and cardiac hypertrophy is favorable. This type of hypertension may become less variable and tend to remain at the higher levels, and the physician must bend every effort to preventing this. Management especially directed toward the relief of emotional strain may prove the most effective agent in obtaining the desired result.

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DIAGNOSES

CASE 16311

Essential hypertension; secondary renal changes.

Arteriosclerotic and hypertensive heart disease.

Bronchial asthma.

CASE 16312

Essential hypertension.

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THE INDEX CATALOGUE

THE War Department at Washington is to be congratulated on its decision to continue beyond the present series the Index Catalogue of the Surgeon General's Library. The Index Catalogue has been of so much worth to the medical world that it is not surprising that William H. Welch once said: "I question whether America has made any larger contribution to medicine." Sir William Osler, speaking of the same publication, remarked, "No undertaking in bibliography of the same magnitude with a special subject has ever been issued." To deprive the world of this Catalogue would have been a very serious loss to the scientific side of medicine.

The history of the Index Catalogue is of considerable interest in view of the fact that a decision has been reached to continue the volumes in the same form as the first editor, Dr. J. S. Billings, began them. In 1880, when the library of the Surgeon General's Office had reached a considerable size, the first volume was issued, listing by authors and subjects all the literature received by the Surgeon General, cumulating for twenty-year periods. The first series consisted of fourteen volumes, published between 1880 and 1895; the second series began the next year and ran to twenty-one volumes, up to the year 1916; in the third series, begun in 1918, seven volumes have appeared, covering about half of the alphabet. The original scheme to list *all* the literature has had to be modified in view of the great increase in medical and scientific articles during the last fifteen years and this has led to some criticism in regard to the value of the third series.

Early this year the whole question was brought forward by the Surgeon General, when it was contemplated that the Catalogue end with the last issue of the third series and a yearly volume, giving medical book-lists only, be substituted. We are fortunate now in having the decision made that the Catalogue will continue in its old form, and it is hoped that it will be even more complete than in the past, for although the *Quarterly Cumulative Index Medicus* gives a yearly summary (not entirely complete), of the important medical literature, the value of a twenty-year cumulation in a single catalogue can hardly be overestimated. As pointed out by the excellent summary of the situation issued by Dr. Archibald Malloch, Librarian of the New York Academy of Medicine, if the new scheme had gone through, "after 1925, to cover another 20-year period of the same subject, one must consult 20 volumes of the *Index Catalogue* for books and theses, plus 40 volumes of the *Quarterly Cumulative Index Medicus* for the periodical literature, requiring 60 times the effort needed now." Apparently the Surgeon General and his advisers saw the value of this argument and the War Department is to continue the world-famous Index Catalogue.

TRIBROMETHYL ALCOHOL (AVERTIN) —ANAESTHETIC

THE art of anaesthesia has been making rapid strides since that memorable day in 1846 when the value of ether as an anaesthetic was first publicly demonstrated at the Massachusetts General Hospital. The latest agent to receive considerable attention is Tribromethyl Alcohol (Avertin). This substance was synthesized in Germany by Willstatter and Duisberg, and first applied clinically by Butzengeiger in March 1926. Since that time it has been used extensively in Europe. J. S. Lundy, of the Mayo Clinic, has undertaken and published a very complete review of its use. This review, in the Proceedings of the Staff Meetings of the Mayo Clinic of December 18, 1929, embraces material from 176 publications on Avertin. Lundy's conclusions should be of real value to surgeons considering the use of this anaesthetic.

There are differences of opinion as to the mode

of application and extent of usefulness of Avertin. It is rather unstable chemically, decomposing to free hydrobromic acid and must be carefully prepared and used only when freshly made. It is tested with Congo Red for neutrality. An aqueous solution of 2½ to 3% is generally used, and given as a rectal instillation. The amount should average 100 mgm. of the drug per kilo body weight, and should not exceed 150 mgm. per kilo. Careful preliminary preparation is advised, using a cleansing enema and some sedative such as morphine or chloretone. Lundy uses both. The entire dose of anaesthetic is usually instilled at one time under slight pressure. The patient drops quietly to sleep in 5 minutes.

Lundy summarizes the *advantages*, as quoted by various authors, as follows:

1. peaceful induction
2. long postoperative sleep.
3. early abdominal relaxation
4. ease of reabsorption of the drug
5. reduction in amount of ether necessary as an adjunct
6. decrease in pulmonary complications
7. lack of production of thrombi or emboli

Disadvantages quoted are:

1. long postoperative sleep
2. excitation during and after anaesthesia
3. singultus
4. ready decomposition of Tribromethyl Alcohol in solution, or its precipitation out of solution
5. obstruction to respiration by falling back of the tongue
6. reduced alkali reserve
7. postoperative bronchopneumonia
8. initial hyperesthesia of skin and early engorgement
9. decrease in concentration of blood calcium
10. irreversibility

The majority of *complications* occur in the first 30 minutes of anaesthesia. Circulatory depression is most common. Respiratory depression is reported.

Avertin has been used in *obstetrics*, but labor pains cease and instrumental delivery is necessary.

Hirsch and Killian reported favorably on its use in *nose and throat work*.

Success with Avertin has been reported in the control of *maniacal patients* and of the spasms in *tetanus*.

There seems to be some real danger in the effect of the drug on the liver, where it is detoxicated, and on the kidney whence it is excreted. Disease of these organs contraindicates its use.

Lundy concludes that the chief use of Tribromethyl Alcohol is as a *basic anaesthetic*. He considers it distinctly dangerous except in careful and intelligent hands, and not so efficient as ether and oil in rectal anaesthesia.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

SHOEMAKER, ROBERT 3D. M.D. University of Pennsylvania 1916. Roentgenologist, The Lankenau Hospital, Philadelphia and The Joseph Price Memorial Hospital, Philadelphia. Address: 68 Byberry Road, Hatboro, Pennsylvania. Associated with him is:

ECKELS, JOHN C. A.B., M.D. Harvard 1927. House Officer, Lankenau Hospital, Philadelphia 1928-1930. Assistant Resident in Urology, Peter Bent Brigham Hospital, 1930-. Address: 721 Huntington Avenue, Boston. They write on: "Bullet in the Heart. Report of Case with Recovery." Page 195.

FITZ HUGH, GREENE. M.D. Harvard 1927. Junior Visiting Physician, Boston City Hospital. Assistant in Medicine, Harvard. His subject is: "A Clinical and Pathological Study of Chronic Myocarditis." Page 201. Address: 264 Beacon Street, Boston.

PALMER, ROBERT STERLING. A.B., M.D. Harvard 1925. Assistant in Medicine, Massachusetts General Hospital and Harvard Medical School. His subject is: "The Treatment of Essential Hypertension." Page 208. Address: 264 Beacon Street, Boston.

TENNEY, BENJAMIN, JR. B.S., M.D. Harvard 1925. Assistant Obstetrician, New England Baptist Hospital, Cambridge Hospital and Massachusetts General Hospital. His subject is: "Postpartum Eclampsia with the Presentation of Two Cases." Page 211. Address: 271 Beacon Street, Boston.

CROTHERS, BRONSON. A.B., M.D. Harvard 1910. Assistant Professor in Pediatrics, Harvard Medical School. Visiting Neurologist to Children's and to Infants' Hospital, Boston. Address: Children's Hospital, Boston. Associated with him is:

COBB, STANLEY. A.B., M.D. Harvard 1914. Bullard Professor of Neuropathology. Visiting Physician for Neurology at the Boston City Hospital. Address: Dept. of Neuropathology, Harvard Medical School, Boston. Their subject is: "Report of a Case of Progressive Athetosis with Lesions in the Basal Ganglia." Page 213.

HAYDEN, E. PARKER. A.B., M.D. College of Physicians and Surgeons, Columbia University 1919. F.A.C.S. Assistant Surgeon to Out-Patients, Massachusetts General Hospital. Surgeon to Out-Patients, Collis P. Huntington Memorial Hospital. Assistant in Surgery, Harvard Medical School. His subject is: "Internal Hemorrhoids — Injection Treatment." Page 218. Address: 270 Commonwealth Avenue, Boston.

HOLMES, ARTHUR D. B.S., Ph.D. Johns Hopkins University, 1911. Teaching Record Lab-

oratory Assistant, Dartmouth College, 1905-1906. Instructor in Chemistry in University of Maine 1906-1907, etc. Research Chemist, U. S. Dept. Agriculture, Dec. 1911-1918. Now Director of Research, The E. L. Patch Company, Stoneham which is his present address. Associated with him is:

PIGOTT, MADELEINE G. B.S. in Chemistry, Tufts College Graduate Work in Medical Sciences, Harvard Medical School. Research Assistant, Harvard Medical School and E. L. Patch Co. Address: E. L. Patch Company, Stoneham, Mass. Their subject is: "The Relative Antirachitic Activity of Cod Liver Oil and Irradiated Ergosterol (Viosterol)." Page 220.

WHITE, JAMES C. A.B., M.D. Harvard 1923. Associate in Surgery, Massachusetts General Hospital. Instructor in Surgery, Harvard Medical School. His subject is: "Progress in the Surgery of the Sympathetic Nervous System." Page 226. Address: Massachusetts General Hospital, Boston.

THE BOSTON MEDICAL LIBRARY

VERTEBRAL OSTEOMYELITIS

THE recognition of osteomyelitis in the long bones is far less often overlooked than it used to be. Before, however, such a statement as this could be made the medical profession had to be trained to realize that the syndrome of an acute septic infection might be derived from an osseous focus and that at the outset, the time when it was of the highest importance to know this, there were few, if any, focalizing symptoms.

Especially perplexing were those cases occurring in the immediate vicinity of a large joint, where confusion with an arthritis was an easy error into which to fall. After a time however medical observers have added this to the list of lesions to be borne in mind when confronted by evidence of profound and unexplained toxemias.

If there is ground for confusion in the diagnosis of this lesion in the long bones there is infinitely more to be said in defense of failure to recognize it in the vertebral bodies and their several processes although the initial clinical picture is essentially the same, except that from the very fact of its localization in the vertebral column anatomical factors enter the picture tending to lead the investigator astray, as is frequently the case where the spine and the nerves which make their exit from it are involved.

The experience of any one observer is not very great for at most the condition is rare but inversely to the frequency of its incidence is its gravity as a clinical emergency. No one should regard it in any other sense. As it has presented itself to the writer it has always been a fulminating process giving the impression of profound sepsis with rather rapid onset, high fever and excruciating pain. In one instance there was a phlegmon in the posterior cervical region and also bulging of the posterior pharyngeal wall with a torticollis.

In another case the lesion was in the last lumbar vertebra and there was a large abscess in the lumbar region which was traced down to its point of emergence from the body of the last lumbar and considerable necrotic bone was scooped away with a curette. Eventually this incision ceased to discharge and for several months the man was free from symptoms but subsequently has been opened in front five or six times to evacuate large psoas abscesses which have invariably closed within a fortnight after they were opened.

This experience has extended over three or four years. The culture from these abscesses has been the staphylococcus in every instance. The writer has observed cases where the condition was mistaken for perinephritic abscess, appendicitis and inflammation in the broad ligament. In all cases a surgical attack was made for the purpose of treating these supposed lesions and it was then that it was discovered that the trouble was in the vertebral bodies. The acute onset of symptoms, the high fever and the evidence of severe toxemia should rule out tuberculosis before operation and at operation bacteriological evidence should be obtainable which ought to establish a diagnosis.

Knowledge of the fact that in spinal lesions reference of pain away from the spine is a common phenomenon should lead to an examination of the spine which the writer's experience leads him to believe would furnish evidence that should serve to focalize the trouble in the vertebral column. This would be in the nature of muscle spasm and consequent limitation of motion.

The appropriate literature serves to emphasize the importance of these diseases to the diagnostician and those interested in this subject may find instructive material in the Medical Library during the fortnight beginning August 4.

MISCELLANY

MAINE MORTALITY STATISTICS: 1929 (PROVISIONAL SUMMARY)

Washington, D. C., July 29, 1930.—The Department of Commerce announces that there were 11,353 deaths in Maine during 1929 as compared with 11,005 in 1928.

No death rates for 1929 have been computed because any rates based on population estimates made at this time would be unreliable and would have to be materially revised as soon as the 1930 census figures become available.

The number of deaths (523) from influenza in 1929 was almost double the number (278) which occurred in 1928, and this increase was mainly responsible for the increase in the total number of deaths which

occurred during the year 1929, as compared with 1928. The number of deaths (2,189) due to diseases of the heart represent the largest number for the four year period. Cancer, with 1,120 deaths, also showed a continuous increase. The increase in the number of deaths from diarrhea and enteritis in children under two years of age was especially noticeable in the two years, 1928 and 1929.

Most significant among the decreases in deaths were those due to nephritis (877 to 825), and cerebral hemorrhage and softening (1,134 to 1,090). Decreases were also reported for a number of the epidemic and infectious diseases, including diphtheria, scarlet fever, typhoid fever, whooping cough and others.

The number of deaths from accidental and unspecified external causes decreased from 638 in 1928 to 585 in 1929. Especially noteworthy was the decrease in the number of drownings from 114 to 81. This decrease, however, was more than offset by the large increase from 115 to 151 in the number of deaths due to automobile accidents and an increase of 20 in the number of suicides.—*Bureau of the Census Division of Vital Statistics, Washington.*

AN ACT OF CONGRESS PROVIDING FOR MEDICAL AND OTHER CARE IN PENAL AND CORRECTIONAL INSTITUTIONS

An Act of the 71st Congress authorizes the United States Public Health Service to supervise and furnish medical and psychiatric and other technical and scientific services in Federal penal and correctional institutions. The surgical, urological, eye, ear, nose and throat, general medical and psychiatric practice in a large modern prison affords a wide choice for young men to specialize in several branches of medical practice. The opportunities which such a service may offer as a stepping stone to a commission in the Regular Corps of the Public Health Service is made possible through recent congressional legislation. Moreover special advantages and future careers for young men with a background and interest in psychiatry are made possible through the recent creation of a Division of Mental Hygiene in the office of the Surgeon General, Bureau of The Public Health Service. Opportunities are also offered for the older man with an ability and experience in the treatment and care of mental diseases to contribute to the sum of knowledge respecting the relationship of this subject to correctional methods and correctional procedure generally. The Surgeon General of the Public Health Service invites correspondence from qualified medical men who desire to become identified with this new and important activity.

DAVID V. BUSH ENJOINED

Washington, D. C., July 14, 1930 (F. T. C.)—David V. Bush, who maintains offices in Chicago, has been ordered by the Federal Trade Commission to stop representing a course in bodily weight reduction as "Nature's method of reducing", or that it is amazing, wonderful or new.

Bush advertised his method in newspapers and magazines and mailed the instructions to persons in various parts of the country who wrote to ask for them and sent the required amount of money.

Among other representations on the part of Bush as prohibited in the Commission's order to cease and desist, are the following: That his method does not involve processes of starving; that the alleged results will be obtained in a few days; or that upon following the instructions customers will find that their fat melts away like magic and that each of them weighs what he should weigh and that each will necessarily become "slim, buoyant, energetic".—*National Better Business Bureau, Inc., Commercial Department.*

DR. LADD AND DR. COOK WILL EXCHANGE POSITIONS

The report is current that Dr. Henry A. Ladd in charge of the U. S. Veterans' Bureau Hospital at Rutland, Mass., and Dr. Richard L. Cook in charge of the Bureau Hospital at Sunmount, Tupper Lake, N. Y., will exchange posts.

Both of these medical officers have given excellent services.

WEEKLY HEALTH INDEX

Telegraphic returns from 64 cities with a total population of twenty-nine million for the week ending July 19 indicate a mortality rate of 11.2 as against a rate of 10.4 for the corresponding week of last year. The highest rate (37.8) appears for Memphis, Tenn., and the lowest (5.4) for Cambridge, Mass. The highest infant mortality rate (201) appears for Atlanta, Ga., and the lowest for Cambridge, Mass., and Kansas City, Kans., which reported no infant mortality.

The annual rate for 64 cities is 13.1 for the twenty-nine weeks of 1930, as against a rate of 14.0 for the corresponding weeks of 1929.

THE MAX PAM METABOLIC UNIT

The opening of the Max Pam Metabolic Unit for Clinical Research in diseases of metabolism at the Michael Reese Hospital in Chicago was announced this week (July 21, 1930) by Alfred C. Meyer, president.

The operation of this unit has been made possible through a bequest of the late Max Pam and the generous coöperation and vision of the trustees of his estate. A capital fund of $150,000 is provided for the maintenance of the unit.

The unit consists of beds for four patients and an adjoining laboratory completely equipped for the investigation of metabolic diseases along physiological and chemical lines. The patients who are under observation will be given all the detailed care that is necessary in carrying out research problems, but at the same time will not feel isolated, because the unit is located in the midst of the hospital and in especially pleasant surroundings. There is no evidence

of the laboratory in the patients' portion of the unit.

"While the Metabolic Unit is in the hospital proper," said Mr. Meyer, "it will work in closest coöperation with the chemical, pathological, bacteriological, and physiological departments of the Nelson Morris Institute for Research which is a part of the Michael Reese group.

"The Max Pam Metabolic Unit will afford the staff of the Michael Reese Hospital a splendid opportunity to study intensively all diseases of metabolism which include among others, diabetes, obesity, goitre and other disorders of the thyroid gland, gout and arthritis. The close affiliation which will exist between this unit and the research and other departments of the hospital will also give the staff occasion to probe the problems of kidney and heart diseases. The plan of operation now outlined calls for team play by the clinical and research staffs and intensive work has already started."

RECTOR COMPANY, PYORRHEA CURISTS, SUBJECT OF FRAUD ORDER

On July 14, 1930, a postal fraud order was issued against the Rector Co. A. J. Major, President; the J.-L. Mfg. Co. and their officers and agents as such at Kansas City, Mo. The business, which was started in September, 1928, consisted in selling a liquid called "Pyro-Kil" (formerly "Pyro-Nox") and a powder dentifrice as an accessory. This treatment was advertised in numerous publications as a cure for pyorrhea. It was claimed that it "will prove a perfect success in the most severe cases of Pyorrhea, Trench Mouth, Sore, Bleeding and Receding Gums, and Loose Teeth".

The advertising also featured a testimonial from an individual who claimed to have been cured of pyorrhea by the treatment. Solicitor Donnelly showed that this testimonial had been given to another, although similar remedy, and that it had been "deliberately misquoted". — *Bulletin National Better Business Bureau.*

HOW UNCLE SAM PROTECTS TRAVELERS

Excerpts from an Article by F. W. Draper, M.D., Assistant Surgeon General United States Public Health Service, Washington, D. C., Member of the Public Health Service and Administration Section, White House Conference on Child Health and Protection

When we start off for a vacation with the children, Uncle Sam is watching over our family's health and safety. It may be reassuring to know that the federal government through the office of the Surgeon General, is constantly on the alert to safeguard our health through the inspection and regulation of the housekeeping details of trains and steamships.

Safeguarding the health of travelers is only one of the many problems which the Surgeon General and his aides are called upon to solve, but it is increasingly important for more and more American families are acquiring the habit of travel. In fact, so many children are traveling these days that the Public Health Service and Administration Section of the White House Conference on Child Health and Protection, of which the Surgeon General is chairman, will include the study of health laws relating to common carriers in its consideration of measures which affect the welfare of children, when the conference meets next November in Washington.

Here are some of the present regulations enforced through the office of the Surgeon General in regard to drinking water on trains and steamers:

> Every six months a common carrier must secure from the United States Public Health Service a certificate approving the source or sources from which it draws its supply of water for drinking and cooking purposes. This certificate is given only after a survey and examination.
>
> In vessels plying between one state and another the piping for water is likewise subject to inspection, lest the drinking water come in contact with that for washing or fire.
>
> One more protection—and a comparatively recent one—is the regulation requiring separate compartments for ice and water in coolers. Though the ice may be made of pure water, there is always the chance that dirty hands may carry it.

Uncle Sam watches over our food supply too, prohibits the serving of spoiled or tainted food, whether cooked or uncooked, and has recently ruled that we must be served with Grade A pasteurized milk or with certified milk.

The law further provides that all table and kitchen utensils, including crockery and glass must be washed in boiling water and suitable cleansing material after they are used, and that refrigerators and food receptacles must be emptied and washed out with hot water and soap not less than once a week.

Personal cleanliness of the employees is secured by the requirement that they wash their hands after the use of the toilet and immediately before beginning service. To make this possible the law directs that a proper lavatory with soap and clean towels shall be provided for the employees and "kept at all times in a clean and sanitary condition," and places the responsibility for this provision upon the person in charge of the dining car or dining room.

The employees in kitchen or dining room are required to undergo a physical examination before entering on their duties, and at any other time when it is deemed desirable, and if they are found to have or are suspected of having a communicable disease, they are immediately relieved from service.

The little basins into which we clean our teeth on

sleeping cars are there by virtue of the law which forbids us the use of the common wash basin for this purpose. The law also forbids the common towel and the common drinking cup.

Travelers suffering from certain communicable diseases cannot be accepted as passengers in the usual way on trains or boats. It is sometimes necessary, however, for these people to travel and the law provides for this contingency by specific and stringent regulations which safeguard both the patient and the public.

The United States Public Health Service has also, at the request of the Department of the Interior, recently taken over the responsibility for sanitary conditions in our National Parks, and now inspects food and water supplies and enforces regulations regarding the disposal of wastes.

By coöperating with Uncle Sam in his efforts to protect the health of our families we can be of immense service in furthering his program on Child Health and Protection.

CORRESPONDENCE

NATIONAL BOARD OF MEDICAL EXAMINERS

225 South Fifteenth Street, Philadelphia, Pa.

To the Editor:

The facts in the following article, submitted to you for free use, are vouched for by the undersigned.

EVERETT S. ELWOOD, *Executive Secretary.*

Philadelphia, Pa.—At the recent Annual Meeting of the National Board, the following officers were elected: Waller S. Leathers, M.D., President, Everett S. Elwood, Executive Secretary, J. S. Rodman, M.D., Medical Secretary.

In addition to the officers, eight new members were elected for terms of six years each. Three of these are representatives of the Federation of State Boards of Medical Examiners in the United States. They are as follows: T. J. Crowe, M.D., Secretary, Board of Medical Examiners for the State of Texas; J. Gurney Taylor, M.D., member of the Wisconsin State Board of Medical Examiners; and J. H. J. Upham, M.D., Dean of the Ohio State University College of Medicine and member of the Ohio State Medical Board.

The remaining five members were elected at large. They are as follows: Charles A. Elliott, M.D., Professor of Medicine, Northwestern University Medical School; William DeB. MacNider, M.D., Professor of Pharmacology, University of North Carolina School of Medicine; Walter W. Palmer, M.D., Professor of Medicine, Columbia University College of Physicians and Surgeons; E. D. Plass, M.D., Professor of Obstetrics and Gynecology, The State University of Iowa College of Medicine; and Charles R. Stockard, M.D., Professor of Anatomy, Cornell University Medical College.

In addition to the new members, Dr. Howard T. Karsner, Professor of Pathology, Western Reserve University, was elected for a second term of six years to succeed himself.

The constitution of the National Board of Medical Examiners was so amended at the Annual Meeting that its membership was increased from twenty-one to twenty-seven. The amendment provides that a total of fifteen memberships shall be representative and twelve elected at large. Six of the fifteen memberships are made up of three Surgeon-Generals of the United States Army, Navy, and Public Health Service, and of an additional number from the Medical Corps of each of these services. Five memberships are made up of representatives from the state boards of medical examiners. Prior to this the state boards have had only three members to represent them. Two of the representative memberships are from the Council on Medical Education and Hospitals of the American Medical Association, and two from the Association of American Medical Colleges. Except the memberships from the Federal Services, the term of office is six years and no member can have more than two consecutive terms.

The reports of the officers of the Board showed an increase of approximately ten per cent. in the number of candidates taking the examinations during the past year as compared with the year previous.

The number of state boards now recognizing the National Board's certificate total forty besides the territories of Hawaii and Porto Rico and the Canal Zone. Partial recognition is also granted the National Board's examinations by England, Scotland, Ireland, and Spain.

Examinations in Parts I and II were scheduled and given in forty-one centers throughout the country; there being a total of 707 candidates registered for Part I and 337 for Part II. Examinations in Part III, the clinical and practical examination, were held in sixteen centers in June and July with approximately 280 registered candidates.

INFORMATION RESPECTING GONORRHEA

Editor, *New England Journal of Medicine.*

Dear Sir:—

As gonorrhea is probably as serious a problem today as it has ever been, might it not be well to take advantage of all possible help?

At the beginning of this year our very able and wide-awake Department of Public Health published a pamphlet entitled "Information for the Patient" which is probably as truthful a statement about gonorrhea as has ever been printed and is within the understanding of all. I find patients are very grateful for the information which it contains. The seriousness of their condition is impressed upon them and furthermore they seem to regain a more or less lost hope and develop a will to get well, which is of considerable importance in bringing about a cure.

I find, however, there are many physicians who are not familiar with the pamphlet. Might it not be worth while to endeavor to bring about its more general distribution among physicians and they, in turn, to see that it not only reached their active cases but also those that might be classed as potential cases. I believe there are people who if the story contained in the pamphlet was fresh in their memory might think twice before exposing themselves to a possible gonorrheal infection.

I extend my congratulations to the Department of Public Health for its able effort to help a bad situation.

Very truly yours,

OSCAR F. COX, JR.

475 Commonwealth Avenue, Boston.

LANTERN SLIDE DEMONSTRATIONS ON THE DIAGNOSIS AND TREATMENT OF DISEASES AND TUMORS OF BONE

July 20, 1930.

To My Colleagues, the Editors of the Medical Journals of the United States and Canada:

If you receive this in time and it is appropriate, will you publish in your journal that there will be a meeting in the ballroom of Belvedere Hotel in Baltimore, Maryland, Monday, Tuesday and Wednesday, September 15, 16 and 17, 1930, beginning Monday morning at ten o'clock and ending Wednesday evening at nine o'clock, daylight saving time. During these days there will be lantern-slide demonstrations, with four lanterns and screens, on the Diagnosis and Treatment of Diseases and Tumors of Bone.

The first day will be devoted to the fundamental and essential knowledge of the benign and malignant lesions of bone, such as osteitis fibrosa, giant-cell tumors, osteomyelitis, sarcoma and so forth. On the second day, the subject will be the different diseases of single bones, such as the lower end of the radius, vertebrae, etc. The third day will be reserved for the presentation of rare lesions of bone difficult to diagnose. Any member of the medical profession attending this meeting may register such a case by addressing Miss Maude Walker, Secretary to Dr. Bloodgood, Surgical Pathological Laboratory, Johns Hopkins Hospital, Baltimore, Md., enclosing the x-ray films or lantern slides of them (if possible the latter) and sections of tissue, if any. Any member of the medical profession interested in the diagnosis and treatment of lesions of bone is invited.

On account of the size of the ballroom the number must be limited to 800.

Those who wish to attend should write the Belvedere Hotel and register, either requesting the usual rates for a single or double room with and without bath, or the special rates for three or more in a room with and without bath, and the special restaurant rates for a club breakfast, luncheon and dinner. You are advised to bring the answer received from the Manager of the Belvedere Hotel with you and present it when you register. For any further details in regard to this demonstration, address your letter to Miss Maude Walker, named above.

I am very anxious that this invitation should reach radiologists, surgeons, pathologists, and internists who are interested in the subject but have only rare opportunities to observe lesions of bone. In three sessions of two or two and one-half hours each, on three days, with four lanterns and a very remarkable and educational motion picture, the subject can be presented in an almost unforgettable way, emphasizing the essentials and fundamentals in the diagnosis and treatment of bone lesions. All cases registered for presentation on Wednesday, will be sent later to Dr. Bowman C. Crowell, Director of Clinical Research of the American College of Surgeons, who is Chairman of the Bone Sarcoma Committee. You should become familiar with this registration of sarcoma of bone, if you are not, because you can register all your cases there and receive the diagnosis of a committee, and you can send for groups of bone tumor cases which have been registered, for personal study.

It is impossible except in the largest clinics, for any radiologist, pathologist, surgeon, or internist, to become familiar with the changing clinical, x-ray and microscopic pictures of diseases and tumors of bone as they come under observation earlier and immediately after the first injury or first symptom, and to learn how to diagnose and treat them in the best way.

Sincerely yours,

JOSEPH C. BLOODGOOD.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
July 26, 1930.

Managing Editor,
The *New England Journal of Medicine:*

In addition to the articles enumerated in our letter of June 28 the following have been accepted:

Aces Laboratory, Inc.
Mercurochrome Suppositories Aces

Cutter Laboratory
Diphtheria Toxoid-Cutter, 45 cc. vial

Hoffmann-LaRoche, Inc.
Synthetic Thyroxine
Ampuls Synthetic Thyroxine-Roche, 1.1 cc.
Solution Synthetic Thyroxine-Roche
Tablets Synthetic Thyroxine-Roche, 1 mg.

Winthrop Chemical Co.
Mesurol
Ampules Emulsion Mesurol, 20 per cent., 1 cc.

Theocin
Tablets Theocin, 1½ grains
Yours truly,
W. A. PUCKNER, *Secretary*,
Council on Pharmacy and Chemistry.

RECENT DEATH

FLYNN—DR. THOMAS FRANCIS FLYNN of Everett, a Fellow of the Massachusetts Medical Society, died at St. Elizabeth's Hospital, Brighton, July 20, 1930, aged 31.

He was a native of Salem, a graduate of Tufts College Medical School in the Class of 1924 and served as house officer at St. Elizabeth's Hospital.

NEWS ITEMS

THE LISTER ORATION—Dr. Harvey Cushing delivered the Lister Oration before a large audience at the Royal College of Surgeons, on the afternoon of July 9, 1930. The address dealt with the relation of the hypothalamic centers to the pituitary and sympathetic nervous system. The theme was that the diencephalic-pituitary mechanism, being the oldest phylogenetically of the forepart of the brain, should be concerned with the oldest and most primitive vegetative mechanisms, such as excretion, cardiac regulation, feeding, and the perpetuation of the species, and it is precisely these functions which are upset when either the pituitary or the hypothalamus is involved in disease. The symptoms of pituitary disease are similar to those of hypothalamic injury, since the pituitary receives a rich innervation from the hypothalamic centers.

Before delivering the oration, Dr. Cushing unveiled a plaque to Sir Anthony Bowlby and spoke of Bowlby's close relations with the American Expeditionary Forces during the War.

The next evening, Dr. Cushing was tendered a dinner by former members of the staff of the Peter Bent Brigham Hospital now resident in England.

DR. F. D. DONOGHUE IS PLANNING FOR THE STUDY OF CANCER—Dr. Francis D. Donoghue of Boston has embarked for Europe with the purpose of studying the work done in Germany in an effort to find more effective methods applicable to dealing with cancer. Dr. Donoghue has long suspected that mesothorium has a useful place in cancer therapy and he is open-minded with respect to the possibility of the existence of a cancer germ. His reports of observation on work now in progress will be of interest.

DR. WILLIAM H. WELCH VISITS CALIFORNIA—That he may make a study of the fifteenth, sixteenth and seventeenth century books in the Harry E. Huntington Library at Pasadena, California, Dr. William H. Welch is visiting there.

DR. FRANK BILLINGS GIVES $100,000 TO ESTABLISH FELLOWSHIPS AT RUSH MEDICAL COLLEGE—The announcement was recently made that Dr. Frank Billings, for many years professor of medicine and dean of the faculty, had given $100,000 to establish four fellowships at Rush Medical College. They are to be named the Edwin R. Le Count, Ernest E. Irons, Wilber E. Post and Rollin T. Woodyatt Fellowships.

SURGEON F. M. EVANS, U. S. P. H. SERVICE, HAS BEEN ASSIGNED TO DUTY IN BOSTON—A. A. Surgeon F. M. Evans has been relieved from duty at Rosebank, N. Y., and assigned to duty at Quarantine Station, Gallops Island, Boston, Mass. July 12, 1930.

NOTICES

DR. H. F MILLER BECOMES AN ASSOCIATE WITH DR. C. E. AYERS

Dr. Charles E. Ayers announces the association with him of Dr. Harold F. Miller at 36 Pleasant street, Worcester, Mass.

WEST MEDWAY NEEDS A DOCTOR

A report has been forwarded to the *Journal* office that there is an opening for a physician in West Medway.

The people of West Medway are reported to be prosperous and the town is an attractive place in which to live.

Dr. Samuel Butler, a Fellow of the Massachusetts Medical Society, lives there and would, we believe, give all desired facts.

Dr. Quint, now deceased, practiced there for many years.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

March-October — International Medical Postgraduate Courses in Berlin. Complete notice appears on page 853, issue of October 24, 1929.

August 13—Greater Boston Medical Society. Complete notice appears on page 194, issue of July 24.

September 8-12—Postgraduate week of Physical Therapy. Detailed notice appears on page 1025, issue of May 22.

September 14-18 — Fifth International Congress on Physiotherapy. Complete notice appears on page 906, issue of October 31, 1929.

September 16-17-18—Clinical Congress of the Connecticut State Medical Society. Complete notice appears on page 194, issue of July 24.

September 26-27—New England Surgical Society. Detailed notice appears on page 1028, issue of May 22.

October 13-17—Clinical Congress of the American College of Surgeons. Complete notice appears on page 1175, issue of June 12.

October 27—The American Public Health Association. Detailed notice appears on page 1271, issue of December 19, 1929.

March 23-27, 1931 — Fifteenth annual clinical session of the American College of Physicians. Detailed notice appears on page 790, issue of April 17.

BOOK REVIEW

La Rachianesthésie. By EMILE FORGUE and ANTOINE BASSET. Masson & Co., Paris. 30 Fr.

This is a paper-bound book of 222 pages. It is quite interesting, covering, as it does, the complete field of spinal anesthesia from the continental viewpoint. It is clear and very methodically written, the various chapters, headings and sub-headings, and many italics clearly indicating the comparative importance of the various subjects presented.

In the chapter on physiology, and in many places throughout the rest of the book, considerable emphasis is laid on the statement that simple spinal puncture with the withdrawal and re-injection of spinal fluid, but without the introduction of any drug or other foreign substance, will cause meningeal irritation with the production of later symptoms as headache or meningismus. The authors are opposed to the use of this anesthesia in operations above the diaphragm, and consent to its use in operations in the upper abdomen only with reservations. Its great field of usefulness, they feel, is in lower abdominal operations. They do not believe in its use in strangulated hernia, because of the possibility of spontaneous reduction. They warn of the danger of spontaneous defecation during hysterectomy, and cite two cases of pelvic soiling with feces, one of them fatal. Its use in paralytic ileus appeals to them.

They consider as contraindications low blood pressure, septicemia, syphilis, and age below fifteenth year. Extreme age is not a contraindication.

Puncture is made in the median line with the patient in the left lateral position, and spinal fluid is evacuated about in proportion to its pressure. The importance of keeping the puncture below the second lumbar vertebra is stressed. Syncain is the drug used, usually in 8 per cent. solution specially prepared. Injection is made very slowly and without barbotage.

The technic employed appears to be extremely cautious. From the American viewpoint the dose of the drug used, namely, from 4 centigrams to an extreme maximum in upper abdominal surgery of 12 centigrams seems quite small. It would be interesting if we could know how frequently with this technic the anesthesia is not of sufficient height or length.

In discussing the mortality they review 222,467 cases and find the total mortality to be 1 in 1,316.

There is an extensive bibliography appended, and throughout the text are profuse references to the literature. In fact a reader gains the impression that he is getting rather more the divergent views of other writers than he is the authoritative statements of the authors themselves.

In some respects the book is disappointing and gives the impression of being out of date. For instance, in the discussion of spinal anesthesia in America the latest information from which deductions are drawn appears to be about 1927. Consequently the conclusion is reached that spinal anesthesia has received but scant attention in America, and is little used here. While some American authors are quoted, none of them are outstanding users of spinal anesthesia. The omission of Babcock is particularly impressive. No mention is made of the use of ephedrine, although at least two authors who recommend ephedrine are represented in the bibliography. Virtually no mention is made by the authors of their own results. The only exception to this appears to be the short statement that Forgue has had over 4500 cases without a death, and, in another part, that one of them has had over 4000 cases with but a few (specified) late complications.

BOOKS RECEIVED FOR REVIEW

Slit-Lamp Microscopy of the Living Eye by Dr. F. Ed. Koby. Translated by Charles Goulden and Clara Lomas Harris. Second Edition. Published by P. Blakiston's Son & Co. Inc. 360 Pages. Price $4.50.

Publications from the Robert Dawson Evans Memorial for Clinical Research and Preventative Medicine—Endocrine Studies.

Medicine in the British Isles by Sir D'Arcy Power. Published by Paul B. Hoeber Inc. 84 Pages. Price $1.50.

The Surgical Clinics of North America, June 1930. Volume 10, No. 3. Published by W. B. Saunders Company. 265 Pages.

Hypertension by Leslie T. Gager. Published by The Williams and Wilkins Company. 158 Pages. Price $3.00.

Thérapeutique Médicale—1—Maladies du Tube Digestif by M. Loeper. Published by Masson et Cie, Editeurs. 372 Pages. Price 50 fr.

Traitement Médical Des Affections Stomacales, by Léon-Meunier. Published by Masson et Cie, Editeurs. 212 Pages. Price 22 fr.

La Diathermie Dans Les Affections du Tube Digestif by Marcel Laemmer. Published by Masson et Cie, Editeurs. 98 Pages. Price 12 fr.

Medical and Surgical Year-Book—Physicians Hospital of Plattsburgh. 1929. Published by The William H. Miner Foundation, Plattsburgh, New York. 322 Pages.

Clio Medica edited by E. B. Krumbhaar. III-Anatomy by George W. Corner. Published by Paul B. Hoeber Inc. 82 Pages. Price $1.50.

Doctor and Patient by Francis W. Peabody. Published by The Macmillan Company. 95 Pages. Price $1.50.

Injuries to Joints by Sir Robert Jones. Third Edition. Published by Humphrey Milford, Oxford University Press. 195 Pages. Price $2.00.